STUDIES ON ROOT-KNOT NEMATODES IN RELATION TO ENVIRONMENTAL POLLUTION

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CERTIFICATE

This is to certify that Mr. Mujeebur Rahman Khan has prepared this dissertation as required for M.Phil (Botany) degree of the Aligarh Muslim University under my supervision and guidance. He is allowed to submit this dissertation for evaluation in partial fulfilment of the requirements for the degree of M.Phil. in Botany.

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INTRODUCTION

Man since his existence is aware of the problems of maintaining himself and his descendents on earth. In early days of the human civilization, supply of sufficient food and shelter were the basic needs. But in modern times, waste and endproducts of modern man's activities are raising serious threats to his existence. 'In the process of modernization, industrialization and urbanization, man has created serious problem of environmental pollution which is disturbing the ecological balance., Insatiable quest of man to improve the quality of his life has resulted in a situation in which his own survival seems to be in danger. For a modern living of world's teeming population, a better and more food, dewelling units and industries are rather essential. This would further aggravate the already existing state of the environmental pollution. The time has reached when the modern world must become more acutely aware of the insidious alterations in the quality of the environment and of the serious consequences of these alterations. 'A huge amount of toxic materials originating from many different kinds of industries and from other human activities are indiscriminately thrown out as wastes into nearest convenient environment (air, water, soil). These toxic wastes in excess, exceeding the normal self-regulating capacity of

atmosphere, lead to pollution. Hence pollution can be defined as "undesirable addition of toxic substances in excess and beyond the self-regulating capacity to atmosphere, which adversely affect human or plant life directly or indirectly".

'The toxic substances, responsible for pollution are termed as "pollutants". Environmental pollution is basically of three types viz. air pollution, water pollution and soil pollution. ' Wood (1968) classified air pollutants basically in two categories based on their origin i.e. primary air pollutants and secondary air pollutants. Primary air pollutants are those that originate at the source in a form toxic to living organisms. Such air pollutants may be in gaseous or particulate forms. Gaseous air pollutants are sulphur dioxide (SO_2), oxides of nitrogen (NOx), hydrogen fluoride (HF), ammonia (NH_3), ethylene (C_2H_6) etc. Particulate air pollutants are coal dust, cement dust, flyash, suspended particulate matter (SPM) etc. The secondary air pollutants originate from the reactions between primary air pollutants that originate from the source e.g. photochemical pollutants like peroxyacetyl nitrate (PAN) and ozone (O_3). Some gaseous air pollutants like SO_2 and NOx in high humid conditions are converted into acids (H_2SO_4 and HNO_3) which fall on the ground in form of 'acid rain' during the atmospheric precipitation (Oden, 1968).)

For the proper and healthy growth of the plants, certain ranges of environmental factors like water, air, light, temperature, humidity, minerals etc. are necessary. Over 90% biomass of green plants is derived from atmosphere. Air plays pivotal role in the quality of atmosphere which in turn has over-bearing effect on the health of the plants, because major part of the plant body remains exposed to air. Therefore, in the vicinity of industries and busy traffic roads where air becomes polluted by release of toxic materials into the atmosphere, there is real possibility of air-quality effects on plants. Plant leaves which are mainly involved in photosynthesis, a process responsible for sustenane of the whole plant body, are much vulnerable to air pollution damage. 'Air pollutants adversely affect plant life directly or indirectly and reduce the yield (Mudd and Kozlowski, 1975). Gaseous air pollutants enter the leaves through stomata by absorption or adsorption and cause injuries directly in the leaf tissue or interfere in biochemical reactions (Pell, 1979).

Particulate air pollutants like soil dust, coal dust, cement dust, flyash, etc. mostly fall and deposit on the leaf surface and block the stomatal cavities forming a thin layer on the leaf surface. This hampers transpiration and checks the transmission of solar radiation (Darley, 1966). The acids (H_2SO_4 and HNO_3)

either directly injure the plant parts or indirectly through soil harm the root system. Air pollutants affecting physiology and biochemistry of the plants, induce visible symptoms like chlorosis, necrosis, early senescence, stunting etc. (Heagle, 1973).

Soil and water pollution can be defined as undesirable accumulation of residue of various organic or inorganic materials in soil or water as a result of man's activities (Cole, 1969). Industrial effluents and waste water are mostly discharged into nearest convenient environment like soil, water etc. causing their pollution. The effluents contain toxic substances which cause harmful effects on plants, animals and microbes. In most of the cases heavy metals have been found to be the major part of the toxic substances. Soil pollution due to heavy metals like Hg, Pb, Co, Cr, etc. have now been well recognized through out the world. Refineries, metal smelters, caustic soda industries, paper mills, power plant discharges, soap factories, fertilizer industries, electroplating units etc. are mainly responsible for heavy metals pollution.

Industrial effluents, domestic waste materials and sewage water are discharged into land and rivers, which may reach to crop fields. The effluents and waste water containing a number of toxic substances when reach in the rhizosphere are absorbed by root and toxic substances

accumulate in different parts of the plant (Westing,1969). The effluents and wastes either directly injure the root when come in the contact or indirectly impair the plant growth due to accumulation in different parts. Major portion of toxic substances of the effluents and wastes is shared by heavy metals. Adverse effects of the industrial effluents containing heavy metals on the crops of beans, pea, wheat, tomato etc. have been demonstrated (Ajmal et al., 1984; Ajmal and Khan, 1984).

Root-knot nematodes (Meloidogyne species) are one of the most devastating group of plant parasitic nematodes, as they have exceedingly wide host range and interact with other plant pathogens synergistically, causing greater damages to an array of plants. Sasser (1977) summarized the occurence of root-knot nematode species in different parts of the world. Meloidogyne incognita (Kofoid and White, 1919) Chitwood, 1949; M.javanica (Trueb, 1885) Chitwood, 1949; M.arenaria (Neal, 1889) Chitwood, 1949 and M.hapla Chitwood, 1949 are most widely distributed species. M.incognita is the commonest and most wide spread and attacks crops outdoors as well as in greenhouses. Many plant species suffer considerably due to its attack (Lamberti, 1979). General estimates for the losses of the vegetables in different regions of the world due to root-knot nematodes varies from 11-25% (Sasser, 1979).

In nature, plants are exposed to a variety of biotic and abiotic pathogens and plants often suffer from more than one disease at a time. Synergistic interaction between biotic pathogens particularly between plant nematodes and other plant pathogens have been recognized on many different crop plants (Powell, 1971). Root-knot nematodes also interact with a number of fungal and bacterial plant pathogens. Interactive effects are generally non-beneficial to the host. Interactions between root-knot nematodes and several root-infecting fungi like species of Fusarium, Rhizoctonia, Pythium, Verticillium, Sclerotium, etc. are well established (Powell, 1971, 1979; Khan, 1984). In nature interactions between biotic plant pathogens and air pollutants (abiotic pathogens) may also develop under specific conditions (Pell, 1979). This type of interaction has received very little attention of all those concerned with the health of the plants (Heagle, 1973). Some efforts made in this direction has shown that air pollutants influence the ability of viruses, fungi and bacteria in causing diseases in plants (Heagle, 1973). Kozlowska (1981) observed the harmful effect of industrial dust on the nematode, Panagrolaimus rigidus. It has been observed that in soyabean plants, exposed to O_3 and SO_2 singly or in combination, the reproduction and development of Heterodera glycine and Paratrichodorus minor was inhibited, while Balanoliamus longicaudatus remained

uneffected. However, reproduction of Pratylenchus penetrans was enhanced, when exposed to SO_2, in comparison to the plants exposed to charcoal filtered air or O_3 (Weber et al., 1976). The presence of P.penetrans attacking the roots enhanced the negative effects of O_3+ SO_2 on the leaf growth and dry-weight of tomato (Shew et al., 1982). Root-knot nematode (M.hapla) infected tobacco plants have been found to be more sensitive to ambient ozone (Bisessar and Plamer, 1984). Discernible effects are expected in multipathogenic situations where abiotic pathogens (pollutants) and biotic pathogens (root-knot nematodes) interact. The impact of air pollution stress on the development of root-knot nematodes on crop plants is yet to be investigated properly.

The use of waste water for irrigation may raise serious problems for plants. Cole et al. (1969) stated that pollutants of this kind increase the prevalence of root-diseases and pre-dispose the plants to pathogenic damage. Cooke (1956) isolated plant pathogenic fungi from sewage and polluted water. Heavy metals are known to inhibit microbial activity (Babich and Stotzky, 1982; Maliszewska et al., 1985). It is believed that poor growth of the plants grown in such polluted soil might be due to reduced activity of the soil microflora which are beneficial to plant growth in addition to the direct

effect of heavy metals on the roots of the plant. Bisessar et al. (1983) studied the interaction of heavy metal soil pollutants like Ni, Cu, Co, etc. and root-knot nematodes. They observed that when nematode infected celery plants were transplanted in polluted soil, there was greater incidence and severity of disease caused by M.hapla. More recently Khan et al. (1986), however, observed adverse effects of mercury on M.incognita. Nevertheless, effects of heavy metal soil pollutants on nematodes causing diseases in plants have not received desired study.

In India, environmental pollution due to various kinds of industries, power plants, refineries and automobiles is quite common. These industries release SO_2, HF, NOx, NH_3, O_3, coal dust, cement dust, fly ash in the form of air pollutants and industrial effluents and waste materials containing heavy metals in the form of soil pollutants. A number of vegetables are grown around the industries i.e. in the environment loaded with different pollutants. So the plants might suffer a great loss in case one or the other disease becomes more aggressive under specified polluted conditions. The manifestation of phenomenon of interaction under such conducive environment must be examined in relation to crop performance.) A Thermal Power Plant is situated at Kasimpur, Aligarh,(U.P.)

about 15 km away from the Aligarh Muslim University Campus within parallel 27°29' and 28°11' north latitude and 27°29' and 78°38' east longitude at 640 ft. above the sea level and is known to cause air pollution damage to vegetation (Gupta, 1981; Ghouse and Khan, 1978, 1983). Effluents of some industries of Aligarh have been shown to contain heavy metals and their adverse effects on growth and productivity of various crops like beans, pea, wheat, tomato etc. have been demonstrated (Ajmal et al., 1984; Ajmal and Khan, 1984).

Pollutants may affect parasitism of root-knot nematode in different ways. Parasitism may be favourably or adversely influenced through a direct effect of the pollutant on the parasite or the effects may be indirect through pollutant induced changes in the host plant or through changes in other aspects of the environment. The main objective of the proposed work is to acertain nature of interaction between root-knot nematode and pollutants (air pollutants, heavy metal soil pollutants) and interactive effects on the host and root-knot nematodes. Whether the interaction between the two is synergistic or antagonistic and the interactive effect is harmful or beneficial to the host or there is no interaction between them are some of the basic questions that are expected to be answered by the proposed investigations.

Vegetables which are most preferred group of host crops of root-knot nematodes are grown around the Thermal Power Plant, Kasimpur. Air pollution due to the Thermal Power Plant has been found to affect the growth and productivity of a number of vegetables grown in the area (Gupta, 1981). So there is real possibility of interaction between air pollutants and root-knot nematodes in such a situation. Therefore, it is planned to investigate this possibility of interaction between air pollutants and root-knot nematodes on some vegetable crops. This would include site studies as well as glasshouse studies. At the same time, experiments will also be undertaken to investigate the possibility of intraction between heavy metal soil pollutants and root-knot nematodes on some vegetable crops mainly in artificial treatments under glasshouse conditions. The following aspects will be studied in the proposed research programme for Ph.D.

1. Collection of meteorological data and base-line data on air pollution around the Thermal Power Plant Kasimpur, Aligarh and demarcation of polluted and unpolluted regions around the plant;
2. Incidence and intensity of root-knot disease on vegetables in polluted and unpolluted regions in natural habitat; and analysis of soil characteristics of samples collected from both regions;

3(a) Impact of air pollutants in ambient air due to coal burning in the Thermal Power Plant on growth performance of some vegetable crops;

(b) Impact of air pollutants on growth performance of some vegetable crops under controlled conditions in glasshouse exposure chambers;

(c) Impact of simulated acid rain of different pH on some vegetable crops under controlled condition;

4(a) Interaction of root-knot nematodes (Meloidogyne species) and air pollutants in ambient air on some vegetable crops;

(b) Interaction of root-knot nematodes and some air pollutants under controlled conditions on some vegetable crops in exposure chambers;

(c) Interaction of root-knot nematodes and simulated acid rain under controlled conditions on some vegetable crops;

5. Effect of soils collected from polluted and unpolluted regions on growth performance of root-knot nematode inoculated vegetable crops;

6. Effect of some heavy metal soil pollutants on growth performance of some vegetable crops;

7. Interaction of some heavy metal soil pollutants and root-knot nematodes on some vegetable crops;

8. Histopathological and anatomical studies of roots and leaves of all the above experiments (1-7).

9. Effects of air pollution and heavy metal soil pollution on morphometrics of root-knot nematodes;

10. Impact of air pollution and root-knot nematodes and their interactive effects on certain biochemical aspects of some vegetable crops.

LITERATURE REVIEW

Air is an important limited natural resource vital to animals and plants. The quality or chemical composition of its minor constituents often varies as a result of the emission of contaminants from man's activities. A huge amount of toxic materials originating from different kinds of industries and from other human activities are released into air, which ultimately impure our atmosphere. The toxic materails which are harmful to plants are ozone (O_3), sulphur dioxide (SO_2), oxides of nitrogen (NOx) etc. More than a century ago, Cameron (1874) observed that factory smoke had SO_2 which caused disease in plants. In recent years it has been estimated that crop losses due to air pollutants may be up to 90% (Heck *et al.*, 1982). Heck (1982) suggested that in conducting agricultural research, air quality-agricultural plant ecosystem inter-relationship must be given consideration. Air pollutant which cause diseases in plants are also termed as pathogens. Different air pollutants injure different economically important crops and cause yield losses significantly in them (Darley and Middleton, 1966; Heggestad, 1968; Smith, 1968; Hepting, 1968). Air pollutants also alter the susceptibility of plants to biotic pathogen like fungi, bacteria etc. (Heagle, 1973).

Air pollutants are grouped in two basic categories, on the basis of their origin, i.e. primary pollutants and secondary pollutants. Primary pollutants emanate from their sources in a form toxic to living organisms. They are further differentiated into gaseous or particulate depending upon their forms. Sulphur dioxide (SO_2), oxides of nitrogen (NOx), hydrogen fluoride (HF), ammonia (NH_3), carbon monoxide (CO) etc. are gaseous air pollutants. Coal dust, cement dust, brick kiln dust, fly ash, soil dust, suspended particulate matter (SPM) etc. are particulate air pollutants. Secondary pollutants are formed by reactions between primary pollutants e.g. photochemical pollutants like ozone (O_3), peroxyacetyle nitrate (PAN), Some gaseous air pollutants are responsible for acid rain. SO_2 and NO_2 in high humid conditions are converted into corresponding acids i.e. H_2SO_4 and HNO_3, and during atmospheric precipitation fall on ground in the form of 'acid rain' (Oden, 1968).

Air Pollutants and Plant Diseases

The first report on disease incited on plants by factory smoke coming out of burning of gases appeared in 1874 (Cameron, 1874). However, study on air pollution effects on vegetation did not start prior to mid-1940's. A new kind of plant injury noticed in Los Angeles area of U.S.A. in 1950 attracted interest and concern of all those

concerned with the health of plants (Darley, 1968). Now, there are numerous reports of air pollutant effects ranging from alternations in plant physiology and biochemistry to visible symptoms of chlorosis, necrosis, early senescence, stunting etc. (Heagle, 1973). Guderian et al. (1960) summarized that as with the pathogenic diseases, the extent and nature of injury or damage is determined by genetic and environmental factors as well by the level and duration of exposure to pollutants. The terms 'damage' and 'injury' are often used interchangeably in air pollution plant diseases studies. The designation "pathogen" is given to the living inducers of disease by many pathologists. But disease induced by abiotic factors e.g. air pollutant, drought, extremes of temperature have many features in common with those induced by biotic pathogens. For this reason, Cowling and Horsfall (1979) suggested to use term 'pathogen' to denote any inducer of disease, irrespective of its living or non-living nature. The injury and its severity due to pollution depends upon the type of air pollutants, their concentrations in the ambient air and exposure periods (Barrett and Benetict, 1970; Brandt and Heck, 1968a and 1968b; Darley and Middleton, 1966).

Gaseous air pollutants (Primary type) - Sources and effects:

Sulphur dioxide (SO_2) :-

Sulphur dioxide, an important air pollutant, also known as "London smog" has been known to cause plant

injuries for more than 100 years. It is emitted principally from the following sources (Wood, 1968):

i) Combustion of coal (6% of the total SO_2 emission).

ii) Production, refining and utilization of petroleums and natural gas (20.7%).

iii) Smelting and refining of ores especially copper, lead, zinc, nickel and iron (7%).

iv) Manufacturing and industrial utilization of sulphuric acid and sulphur.

Sulphur dioxide concentration varies from place to place depending upon the amount and concentration of emissions, distance from source, meteorological and topographic conditions. Its concentration is generally inversely proportional to the distance from source and wind velocity. Sulphur dioxide concentration near point sources such as coal burning power plants, smelters etc. with no or little pollution control equipments may be as high as 1 to 3 ppm. In large urban areas SO_2 concentration may vary from 0.05 to 0.40 ppm (Heagle, 1973).

Sulphur dioxide induces the development of several symptoms on plants and plant parts. In leaves its entry occurs through stomata. After entering mesophyll cells of the leaves, it reacts with water to produce sulphite ion which is slowly oxidized to sulphate ion. The sulphate ion

may be utilized by the plant and converted to organic forms (Thomas et al., 1944). The sulphite and sulphate ions are toxic to plant cells when present in excess. The sulphite ions are, however, about 30 times more toxic than sulphate ions (Thomas et al., 1943). First case of SO_2 injury to plant in U.S.A. was recorded in 1905 by Haywood (Haywood, 1905). Barrett and Benedict (1970) gave an illustrated account of the symptoms induced by SO_2. According to them two types of markings (symptoms) viz. chronic and acute appear on leaves due to accumulation of sulphite ions. The chronic markings are generally chlorotic in appearance. Mild chlorosis, yellowing of leaves, and silvering or bronzing on abaxial side are the main chronic symptoms. In some plants red, brown or black coloured patches also appear. Marginal or inter-costal areas of dead tissue are the acute symtoms. These areas at first show a greyish green water soaked appearance but on drying become bleached ivory in colour. The dead or necrotic areas after a short span of time may fall out,leaving a ragged appearance of leaf. When major portion of leaf is so injured, leaf is shed due to formation of abscission layer at the base of petiole. Low concentrations of SO_2 produce chlorosis of leaves without necrotic lesions and veins characteristically remain green (Darley and Middleton 1966; Agrios, 1978).

As SO_2 enters through stomata, day time exposure cause injury to plants as the stomata of most of the plants remain

closed during the night (Katz, 1939; Thomas, 1951). Moisture stress greatly decrease the sensitivity of plants to SO_2 because under such condition stomata also remain closed (Zimmerman, 1952).

Generally, net photosynthesis is reduced in all the plants even at low concentrations of SO_2. However, dark respiration and transpiration rates increase in both short and long exposure durations (Black and Unsworth, 1979; McLaughlim et al., 1979; Takomoto and Noble, 1982; Saxe, 1983a and 1983b). Plants generally show rapid recovery of these processes after termination of exposure lasting upto several days. Several workers have observed increased enzyme activity in some plants exposed to low levels of SO_2 and decreased activity at high concentration of SO_2. Plant metabolism is affected by SO_2 in various ways. SO_2 increases enzymatic activity in some plants at lower concentration and decreases at high concentration (Horsman and Wellburn, 1977; Soldatini and Ziegler,1979; Wyss and Brunold, 1980; Pierre and Queiroz, 1982; Tanaka et al., 1982). SO_2 stimulates phosphorus metabolism (Plesnicar, 1983) and reduces foliar chlorophyll concentration (Pandey and Rao, 1978; Lauenroth and Dodd, 1981). Carbohydrate levels increase in low doses of SO_2, while reduced in high concentration (Koziol and Jardon, 1978).

Due to SO_2 effects on physiology and biochemistry of plant, the growth, development and productivity of the plants are significantly affected. The effects of SO_2 have been studied by several workers both on plants in glasshouse and in ambient air. In open top polythene chambers when wheat plants were exposed to 0.8 ppm SO_2 two hours daily for 60 days, although no chlorosis or necrosis appeared at any stage of the growth, reduction in root and shoot length, number and area of leaves per plant, biomass, number of grains per spike was observed (Pandey and Rao, 1978). Wheat plants were less tolerant to 0.1 to 0.6 ppm of SO_2 doses than maize plants upto 100 h. Different cultivars of wheat were sensitive to a varying degree (Laurence, 1979). There was a significant reduction in the yield of soybean, when plants were exposed 0.09-0.79 ppm concentration in open air fumigation chamber. But no visible symptoms appeared (Sprugel et al., 1980). In groundnut, exposed to SO_2 at 0.06-1.00 ppm concentration four hours daily for six weeks, necrosis and reduction in net primary productivity were observed at 0.25 ppm. Below 0.25 ppm, it was slightly benifical to the plant productivity. An increase in sulphur contents of the plant and reduction in nitrogen and phosphorus contents were also observed (Mishra, 1980). By exposing alfalfa plants to 0.03 ppm of SO_2 concentration continuously, a slight increase in biomass was recorded by Lockyer and Cowling (1981). Saxe (1983b) observed adverse

effects on all plant parts of snap-bean exposed to above 0.1 ppm concentration of SO_2 for 12 h daily for five days in a week. Mejestrik (1980) reported that tobacco plants were found to be more tolerant than cucumber to 0.02 ppm concentration of SO_2 continuously for four weeks. Roots were more affected than shoots of the plants. Flowering was also significantly reduced. Lostein et al. (1983) exposed tomato plants to 0.12 ppm concentration 72 h per weeks. They observed slight decrease in ascorbic acid of ripe fruit while soluble and total solid contents remained unaffected.

Oxides of nitrogen (NOx):-

Nitric oxide (NO), nitrogen dioxide (NO_2) and nitrogen trioxide (N_2O_4) have been recently recognized as important primary air pollutants. They are produced primarily during high temperature combustion (Taylor and MaClean, 1970). Nitrogen oxides are produced during the following processes:

i) Gasoline combustion in automobiles, diesel railway engines and aeroplanes

ii) Petroleum refining

iii) Combustion of natural gas, and coal

iv) Incineration of organic wastes

v) Manufacturing of sulphuric acid by Chamber's process and nitric acid

vi) Manufacturing of paints, roofing, rubber, soap and nylon intermediates

Oxides of nitrogen affect vegetation adversely, reducing plant growth rate (Taylor and Eaton, 1966; Capron and Mansfield, 1977), photosynthetic rate (Hill and Bennett, 1970; Bull and Mansfield, 1974) and fruit yield (Thompson et al., 1970; Spierings, 1971). There is direct evidence that NO_2 - derived nitrogen is absorbed by exposed plants and translocated throughout plant tissues (MaClean et al., 1968; Agrios, 1978; Rogers et al., 1979; Yoneyama et al., 1980). There is no report of appearance of visible symptoms due to nitric oxide (NO) (Taylor and MaClean, 1970). Acute folair symptoms that appear on the plants exposed to high concentrations of NO_2, include water soaked lesions followed by rapid tissue collapse. The lesions later extend throughout the leaf to form white to tan or brown coloured irregular necrotic patches. The interveinal lesions are found to be prominent at apex and along the margins of leaf (Benedict and Breen, 1955a and 1955b; Middleton et al., 1958; Taylor and Eaton, 1966; MaClean et al., 1968). MaClean et al. (1968) observed, besides foliar symptoms, high concentrations of NO_2 causing abscission of leaves and fruits of citrus and hibiscus.

In ambient condition NO_2 affects plant growth, even at the concentration below 1.0 ppm (Taylor and Eaton,1966). But Rogers et al. (1979) observed that NO_2 at 0.097 ppm, 0.152 ppm or 0.325 ppm concentrations in ambient air were not effective significantly on beans.

Fluorides:-

Fluoride containing compounds such as hydrogen fluoride (HF), and silicon tetrafluoride (SiF_4) are important air pollutants (Heagle, 1973). Fluoride is wide-spread in the earth crust as a natural component of soil, rocks and minerals (MacIntire, 1945). Air pollutants containing fluoride are released from the following sources (Treshow and Pack, 1970; Heagle, 1973).

i) Aluminium reduction processes
ii) Manufacturing of phosphate fertilizers and steel
iii) Pottery and ferro enamel works
iv) Refineries
v) Rocket fuel combustion

Fluorides are toxic to plants at much lower concentrations than most of other air pollutants. They enter through the stomata and are rapidly translocated to leaf tips and margins (Jacobson et al., 1966). Fluorides act both as a cumulative poision on some plants and accumulative on others (Heggested, 1968). The characteristic

symptom of hydrogen fluoride is turning the leaf margins of dicots and leaf tips of monocots light to dark brown. Actively grown leaves are more sensitive to fluorides (Agrios, 1978). Fluorides also produce symptoms on leaves, flowers and fruits. Necrotic and chlorotic lesions may occur. The lesions may be grey or light green at first, but later on become reddish brown to tan in colour. Abscission of leaves is also induced by fluorides (Heggestad, 1968). Fluorides also cause necrosis and chlorosis in sepals and petals. In some cases, fruits are more sensitive than leaves, resulting in premature ripening (Treshow and Pack, 1970). Among the plant species most sensitive to fluoride injury are certain varieties of gladiolus, apricots, prunes, peaches, corn,grapes, tulip, while celery, alfalfa, tomato, tobacco are resistant to fluoride (Zimmerman and Hitchcock, 1956).

Ammonia (NH_3):-

Ammonia originates from ammonia fertilizers, brick-kiln plants, ceramic and pottery industries, refineries, cooling plants in cold stores etc. Field injury from gaseous ammonia has been reported during the use of anhydrous ammonium fertilizers. NH_3 causes acute tissue collapse in the leaves with or without subsequent loss of chlorophyll. Leaves show a cooked green appearance, becoming brown or remaining green on drying. The necrotic

lesions along the leaf margins have been reported in sensitive species. Benedict and Breen (1955a and 1955b) observed extensive and widespread injury within 1-2 miles of the ammonia spill. Complete collapse of leaf tissue occured near the source. Several sensitive species showed blackened tissue at distances upto a mile from the spill. Necrotic areas, bright tan in colour, developed in several cereals and grasses. Necrotic and chlorotic interveinal streakings also appeared on plants at some distance from spill. Some other plants produced glazing with or without scattered necrotic spots on the upper surface. Several varieties of peach and apple fruits were adversely affected by ammonia released in the storage houses. Peaches became black in colour at the concentration of 400 ppm of NH_3 (Brennan et al., 1962). Thornton and Setterstron (1940) observed significant injuries in black wheat, coleus, sunflower and tomato foliage when they were exposed to NH_3 at 40 ppm concentration for 1 h. However, NH_3 was also effective in inducing slight marginal injuries in leaves.

Other air pollutants:-

This group may include the pollutants like ethylene, chlorine, hydrochloric gas, hydrogen sulphide, etc. which are regarded as considerably toxic to plants but their effects have not been well recognized as yet. These pollutants originate from refineries, chemical and plastic

industries, glass-making factories, incineration, scrap-burnings, accidental spillage, combustion of petroleums etc.

Ethylene (C_2H_6) has been found to be effective even at very low concentration. Davidson (1949) and Darley et al. (1966) observed injury in orchids at 2 ppm for 24 h and 0.1 ppm for 6 h. Wallace (1927) reported that high levels of ethylene cause epinasty, chlorosis, necrosis, leaves and buds abscission and failure of lower buds to open. Heck et al. (1970) observed that older leaves are more sensitive to ethylene than the youngers. Heck et al. (1962) summarized that cotton, cowpea, black berry, roses, etc. are most sensitive to ethylene and suffer considerably.

Chlorine effects on plants are confined to the limited areas, because it originates from the selected sources. In the ambient condition, chlorine is reported to cause browning and drooping of the leave of sugar maple and crab apple; marginal injuries in horse chestnut; intercostal markings in virginia creeper and blackberry and bleaching of lower surface of older and middle-aged leaves (Heck et al., 1970). Brennan et al. (1965) reported relative sensitivity of 26 plants species to chlorine. Among them radish and alfalfa were the most sensitive at 0.10 ppm for 2 h. Petunia and pinto bean required 0.80 ppm for 4 h to produce symptoms of same severity. It is

generally recognized that like ethylene, older leaves are more sensitive to chlorine than the youngers. Mustard, chickweed, sunflower, radish and alfalfa are the most sensitive plants to chlorine. Chlorine is found to be less effective in inducing injuries under low soil moisture (Benedict and Breen, 1955a and 1955b).

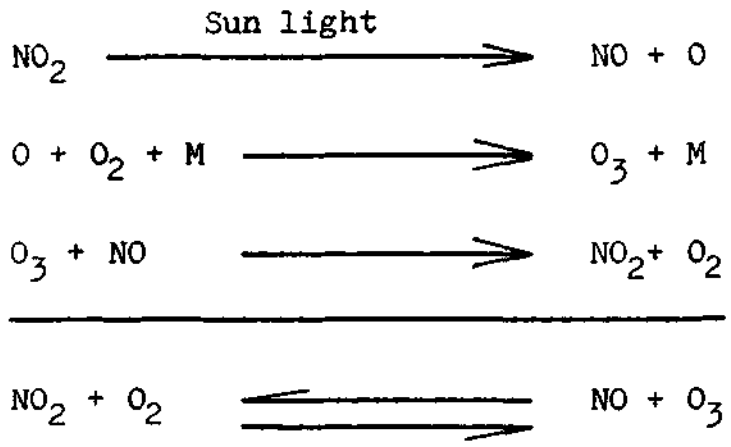
Hydrogen chloride gas is another air pollutant whose effects are also not studied properly. HCl gas is mostly found in high concentrations in the areas of chlorine manufacturing plants and rocket launcher pads. Most of the test plants showed the similar effects like that of NO_2 at the concentrations of 5-10 ppm for 15-60 min (Heck et al., 1980). Swieki et al. (1982) reported that bean leaves with their cuticular wax were more sensitive to HCl.

Gaseous air pollutants (Secondary types) - Sources and effects:

Ozone (O_3):-

It is a typical a secondary type of pollutant formed by reaction between primary pollutants in the presence of ultraviolet light (sun light) and hence called as "photo-chemical pollutant". Ozone is a major constituent of "Los Angeles smog". The most important source of ozone is the photochemical reactions in polluted atmosphere.

Oxides of nitrogen emitted by petroleum combustion (automobiles industries and utilities) react in the presence of light (ultraviolet light) with oxygen to from ozone (Leighton, 1961), as indicated in the following equation, were M is energy absorber:



When incompletely burned unsaturated hydrocarbons are also present in the polluted atmosphere, they are oxidised and this process facilitate conversion of nitric oxide into nitrogen oxide, in such condition, the ozone level reaches upto 1 ppm (Wood, 1968). In ambient air high ozone concentrations (beyond permissible limit) are found in metropolitan cities mainly due to automobile exhausts.

Ozone has been recognized as most destructive secondary air pollutant. Specific damage to an agricultural crop caused by ozone was first reported on grapes by Richards et al. (1958). A year later, Heggestad and Middleton (1959) documented that the cause of weather fleck, a leaf spot problem on cigar-wrapper tobacco in eastern

U.S.A. was due to ozone.

Like the other air pollutants, ozone also enters through stomata and gets accumulated in palisade cells and cause bleaching or discolouration of the palisade tissue which ultimately results into collapse of cells (Pell, 1979). Expanding middle-aged leaves are more sensitive to ozone. The foliar symptoms which O_3 produces include stippling, mottling and chlorosis of usually upper leaf surface. Colour of symptoms varies from light tan to red colour or almost black depending upon the sensitivity of the plant. In some plants viz., citrus, grapes, pines etc. the leaves fall prematurely (Darley and Middleton, 1966; Agrois, 1978). Many deciduous trees, shrubs and some herbaceous plants produce localized thickening of cell walls resulting in dot-like coloured lesions due to ozone (Ledbetter et al., 1959). Generally, the interveinal portions of leaves are injured resulting in angular shaped lesions. Usually veins are not affected except in plants where pigment formation takes place (Heck et al., 1970). Mostly woody and herbaceous plants exhibit bleaching of upper surface of leaves (Ledbetter et al., 1959). In the case of chronic injuries, the epidermal cells collapse and become colourless. According to Heck et al. (1970) leaves often develop water-soaked areas followed by drying and bleaching which result into necrosis on the both surfaces

of leaf. Ozone characteristically injures only palisade and mesophyll cells, while the epidermal cells remain unaffected (Pell, 1979). Many injured cells remain alive, however, their chloroplasts are disrupted and chlorophyll contents significantly reduced (Hill et al., 1961). Ozone produces large light green chlorotic areas with many irregular islands dispersed in them on alfalfa leaves. However, in some plants, tissue eventually becomes uniformly chlorotic and ultimately leaves fall down (Ledbetter et al., 1959).

As ozone enters through stomata, degree of sensitivity of plants to ozone depends upon the density and type of stomata present on leaf. In the susceptible plants, during the exposure, the stomata remains open, while in resistant cultivar, a partial closure of stomata has been observed (Bulter and Tibbitts, 1979). It has been found that the stomata of resistant cultivars of bean are closed at even low concentration of ozone (Faensen-Thiebes, 1983). Engle and Gebelmam (1966) observed that stomata of resistant onions close even at low concentration of ozone. They suggested that membrane of guard cells lose their permeability and leak, thereby closing stomata. The guard cells recovered as soon as O_3 was removed. However, in azalea, sweet corn, soybean, and tobacco no relationship was found between ozone sensitivity and the number of stomata or rate of gas exchange (Gesalman and Davis, 1978;

Harris and Heath, 1981; Feansen-Thiebes, 1983). Environmental factors are also reported to play a major role in the sensitivity of ozone to plants (Ting and Dugger Jr., 1968; Agrios, 1978).

The biochemistry of the leaf is also altered by ozone. Todd (1958) and Todd and Probst (1963) measured the effect of O_3 at 4 ppm for 40 min. on photosynthesis. They concluded that the symptoms were associated with inhibition of CO_2 fixation in leaf. However, Hill and Littlefield (1969) observed decrease in photosynthesis at 0.06 ppm concentration of ozone for 1 h. A significant increase in respiration of pintobean leaves was observed at 4 ppm for 40 min by Todd (1958) and Macdowall (1965). They found that during the first hour of ozone exposure at 0.7 ppm, before the appearance of visible symptoms, the respiration was decreased while after the appearance of symptoms, it was enhanced. Some other workers observed decrease in rate of respiration, when plants were exposed to 0.06 ppm concentration of ozone for one hour (Hill and Littlefield, 1969).

The adverse effects of ozone on growth, biomass and productivity have been studied by several workers in ambient and glasshouse conditions. Blum et al. (1982) observed that when clover plants were exposed to O_3 of 0.06 ppm and 0.09 ppm concentration for 7 h per day for

two years in field condition there was 14% and 27% reduction in forage growth respectively during the second year, while there was 42% root and 24% shoot reduction in clover when they were exposed at 0.05 ppm, 0.10 ppm and 0.15 ppm for 6 days after 32 days of seeding. When soybean plants were exposed to 0.022 ppm and 0.112 ppm for 7 h per day, there was a reduction of 39% in yield and 12.6% in oil contents of the seeds. However, protein contents of seeds remained unaffected (Greenwald and Endress 1984). Pell et al. (1980) exposed potato tuber plants at 0.2 ppm ozone concentration in greenhouse and found reduction in tuber weight while increase in sugars present in tuber was noticed. Similarly, Clarke et al. (1983) observed 60% leaf injuries and 25% reduction in yield of potato tuber when they were exposed to O_3 at 0.3 ppm concentration in ambient condition. Bennett and Oshima (1976) reported 32% and 46% reduction in root dry matter of carrot cv. Inperator-58 at 0.19 ppm and 0.25 ppm of O_3 concentration for 6 h respectively. In cotton cv. Acata SJ-2, there was significant reduction in biomass and boll production, when plants were exposed at 0.20 ppm for 6 h twice a week (Oshima et al., 1979). Ozone caused reduction in root, stem and leaf dry matter of pepper. When pepper plants were exposed at 0.12 ppm and 0.20 ppm concentration for 3 h twice a week, the reduction was 6% and 54% in fruit dry weight respectively (Bennett et al., 1979).

Ozone has been found to be very toxic to tomato plants. There was about 85% reduction in fruit size and 50% in yield when exposed to 0.10 ppm for 20 h (Oshima et al., 1977a and 1977b). Manning and Feder (1976) recorded that O_3 was responsible for 86% fruit number and 91% fruit weight reduction in tomato cv. Tiny Tim, at 0.08-0.10 ppm concentration for 5 h per day once in a week for 5 days.

Peroxyacetyl nitrate (PAN):-

It is another photochemical secondary air pollutant, an important constituent of photochemical smog. Peroxyacetyl nitrate is formed due to reaction between oxides of nitrogen and unsaturated hydrocarbons, released mainly through automobile exhausts, in the presence of sunlight (ultraviolet light) (Wood, 1968; Heggestad, 1968; Agrios, 1978).

$$\text{Olefin} + \text{NOx} \xrightarrow{\text{Sun light}} CH_3\,\overset{O}{\overset{\|}{C}}OONO_2$$

PAN is especially found around metropolitan areas where huge amounts of unburned and unsaturated hydrocarbons and oxides of nitrogen are released through automobile exhausts. This type of pollution is very common in big cities like Los Angeles and New Jersey of U.S.A., where the atmospheric conditions are conducive to inversion layer formation (Heggestad, 1968; Wood, 1968).

PAN also has been found as the most injurious air pollutant for plants. First report on this pollutant causing disease to plants appeared in 1940, when Middleton et al. (1950) observed "silvering" on the lower leaf surface of many herbaceous crops. The characteristic symptoms that PAN produces on susceptible plants include glazing (silvering) and bronzing of lower leaf surfaces and tendency to produce transverse banding on individuals leaves (Bobrov, 1955; Taylor et al., 1960). Younger leaves are injured at their tips and older ones near the base (Agrios, 1978). Transverse banding on individual leaves is related to physiologic age of tissue. PAN causes injury first to mesophyll cells near stomata. This injury separates lower epidermis and mosophyll cells slightly from each other resulting in glazing and bronzing of lower leaf surface. In acute injuries, entire leaf becomes necrotic (Glater et al., 1962). PAN is reported to cause various types of damage in a number of crops like romiane, lettuce, swiss chard, pintobeans, petunia, tomato, african violet, when they were exposed at 15-20 ppb concentrations for 4 h. However, corn, onion, begonia and cotton are usually not injured at 75 to 100 ppb concentration for 2 h (Brandt and Heck, 1968a and 1968b; Darley et al., 1966; Noble, 1965; Heck et al., 1970).

Dugger et al. (1962) found that younger leaves are most sensitive to PAN, but Taylor and MaClean (1970)

observed several day old leaves of pinto beans most susceptible to PAN. Peroxyacetyl nitrate is known to inhibit photosynthesis. Taylor et al. (1983) reported reduction in net photosynthesis in three lichen species when exposed to 0.05 ppm and 0.10 ppm concentration of PAN. Sigal and Taylor (1979) observed reduction in yield of lettuce and swiss chard out of the eight crops they tested at long term intemittent exposure of 0.04 ppm concentration of PAN.

Pollutant mixture:-

There are some reports which show that the presence of SO_2 in low concentration decreases the injury to plant caused by other pollutant. Thomas et al. (1952) reported that the plants receiving Los Angeles air were less injured than those received same air but passed through water. Los Angles air had 0.01 to 0.24 ppm SO_2 while in water passed air no SO_2 was present. Haagen-Smith et al. (1952) observed that plants exposed to pollutants with 0.1 ppm SO_2 had no injury. But injury occured when SO_2 was absent. Hitchcock et al. (1962) observed reduced effect of HF to gladiolus in the presence of hydrocarbons. Menser and Heggestad (1966), however, were first to observe synergistic effect SO_2 and O_3 on tobacco. Since then, there are numerous reports on the effect of mixed pollutants which suggest

that combined effect of pollutants may be synergistic, antagonistic or additive.

Response of plants to pollutant mixtures includes visible symptoms of injury, altered growth and development, physiological and metabolic imbalances and accumulation of certain elements and metabolites. Most important pollutant mixture are O_3+SO_2, SO_2+NO_2 and O_3+SO_2+NO_2. O_3+NO_2, SO_2+HF, SO_2+NaF, NO_2+HF, O_3+H_2S and O_3+acid rain are also known to cause injuries in plants (Reinert, 1984; Heck et al., 1986).

Shew et al. (1982) when exposed tomato plants to 0.2 ppm O_3, 0.2 or 0.8 ppm SO_2 singly and in combination 15 times for 3 or 4 h observed additive effect at 0.2 ppm SO_2 and antagonistic at 0.8 ppm SO_2. When lettuce and radish were treated with 0.4 ppm O_3, 0.8 ppm SO_2 mixture for 6 h there was antagnostic effect in lettuce and additive effect in radish (Ormrod et al., 1983). In a study Shertz, et al. (1980) found increase in leaf abscission and decrease in foliar injury in grape cultivars when exposed to 0.20 and 0.40 ppm O_3 and 0.15 and 0.30 ppm SO_2 mixture for 4 h. Foster et al. (1983) observed significant additive effect in the reduction of growth and yield of potato when exposed to O_3 with 0.1 ppm SO_2 for 6 h daily. Soybean plants exhibited additive, antagonistic or synergistic changes in foliar injury and in reduction of shoot fresh

weight when exposed to 0.25-1.0 ppm O_3 and 0.50-1.5 ppm SO_2 mixtures (Heagle and Johnston, 1979). Olszyk and Tibbits (1982) recorded reduced foliar injury and unaffected leaf area, chlorophyll, leaf weight in pea exposed to 0.06 and 0.27 ppm O_3 - 0.11 and 1.72 ppm SO_2 mixture. Synergistic response in stomatal conductance, antagonistic in foliar injury and additive in the growth was found in snap-bean by Beckerson and Hofstra (1979) and Miller and Davis (1981). Mairie and Ormrod (1984) observed decrease in fresh and dry weight of tomato leaf and root, when exposed to 0.05 ppm SO_2, NO_2 mixture, however, no effect was observed when tomato plants were exposed to these pollutants singly. In soybean cultivars receiving different treatments of SO_2-NO_2 mixture, the synergistic response was observed in photosynthesis, stomatal conductance, chlorophyll reduction; additive in respiration; and reduction in yield at higher SO_2 concentrations (Carlson, 1983; Amudson, 1983; Irving and Millor, 1984).

Acid rain :-

Air pollutants like sulphur dioxide (SO_2) and oxides of nitrogen (NOx) in high humid conditions by reacting with atmospheric water are converted into corresponding acids (H_2SO_4 and HNO_3), which fall on the ground in the form of "acid rain" during the rain fall, (Oden, 1968). Phenomenon of rain fall acidification by air pollutant emmissions was

first noticed in 1757 by Hales in England. However, a century ago, Robert Angus Smith (1870) was first to observe its effects.(Cowling, 1982). Modern attention to acid rain was focussed since 1948 (Oden, 1968). Rain and snow in northern Europe and in the north eastern United States have become progressively more acidic during the past two decades (Oden, 1968; Cogbill and Likens, 1974; Likens and Bormann, 1974). Individual rain fall in Sweden, Norway and eastern United States produced rain of pH 3.0 to 3.6. The average acidity of rainfall in eastern U.S.A. was estimated to be below pH 4.5 in 1972-73 (Cogbill and Likens, 1974). Sulphuric acid has been recognized as a major component of the acid substances in the precipitation both in Europe and North America (Cogbill and Likens, 1974; Likens and Bormann, 1974).

There are a few reports about the direct or indirect effect of acid rain on terrestrial vegetation. The most striking adverse effect was reported on development of peat moss (Sphagnum sp.), an aquatic plant in lakes and streams in Sweden, receiving large amounts of acidic precipitation (Grahn et al., 1974).

Acid rain causes primarily, the acidification and alteration of water and soil. It has been recognized that herbaceous plants are more vulnerable for direct acid rain injury than the woody plants (Heck et al., 1986). Direct

injury of terrestrial plants by artificial mists of simulated rain containing dilute sulphuric acid, appeared in the form of increased leaching of nutrients from pinto bean and sugar maple seedling foliage (Wood and Bormann, 1974). Soybean and kidney bean plants when exposed to acid rain of pH 3.2 and pH 6.0 for 17 weeks, intermittently in the field conditions, no effect occurred on number of pods. Shriner and Johnston (1981) observed no significant effect of acidic soil on fresh weight of shoot, root or pod of soybean, both in field and glasshouse.

Particulate air pollutants:

Particulates have been recognized a plant-pathogenic air pollutants for many years, but relatively little attention has been given to the problem (Wood, 1968). It may be because of the fact that particulates are not toxic to plants unless they are high causitic in nature or in high concentration (Heck et al., 1970). According to Wood (1968) important sources of particulate air pollutants are as follows:

i) Combustion of coal, gasoline and fuel oil
ii) Cement production
iii) Lime kiln operations
iv) Incineration and soil erosion
v) Burning of plants and plant materials and wrong agricultural practices

vi) Volcanic erruption

vii) Transportation

viii) Construction

Major particulate air pollutants are coal dust, lime dust, cement dust, soil dust particles, fly ashes etc. The particulate air pollutants are major problems in developing countries but are not so important and serious in developed countries (Das, 1986). Particulate matters adversely affect a variety of crops resulting in poor growth (Heck et al., 1970). Dust from different sources settle on leaves, flowers either as such or in combination with rain drops or mist to form thick crust (Agrios, 1978). Cement dust is alkaline in nature and contains calcium silicate, which further helps in encrustation of dust on leaves in close vicinity of cement industry (Darley, 1966). According to Agrios (1978), the affected plants may become chlorotic, necrotic, grow poorly and even may die. Additional damage to plants is caused by the toxicity of some of the deposits on leaf tissue either directly or after formation of toxic solutions in the presence of free moisture on the plant. Usually symptoms appear on plants when heavy deposition of particulates occurs. Many particulates are byproducts of agricultural practices and usually inert (Darley and Middleton, 1966; Heck et al., 1970). Colwill et al. (1979) observed black deposits on the leaves of the plants grown along the road-side with highly busy traffic. Such plants

showed poor growth. There have been numerous reports that dust of varying origin interfere with stomatal functioning mostly by filling and blocking the stomatal aperture (Ricks and Williams, 1974; Fluckiger et al., 1978 and 1979) increase leaf temperature (Eller, 1977; Fluckiger et al., 1978) and transpiration (Beasley, 1942; Eveling, 1969); reduce photosynthesis (Darley, 1966) and increase the uptake of gaseous air pollutants (Ricks and Williams, 1974). All these effects eventually result into poor growth of the suffering plants. Singh and Rao (1981) studied in a closed chamber the effect of dust on wheat plants and observed reduction in transpiration, chlorophyll contents and productivity. Lime dust particles form incrustations on leaves of vegetables with a resultant reduction in photosynthesis, vigour and hardness of the plants Heck et al. (1970) summarized that particulate emissions from different sources cause reduction in quality of vegetables and fruits.

Soil Pollutants and Plant Diseases

Heavy metal soil pollutants - Sources and effects:

Soil and water have been used since long as a most cheap and convenient place to dump the industrial effluent and waste materials. These effluents and wastes contain toxic materials which adversely affect plant life

(Ajmal and Khan, 1984; Ajmal et al., 1984). Soil and water pollution have received considerably less attention, while their adverse effects on plants are as severe as of air pollution. According to Cole (1969) soil and water pollutants are produced or released from the following sources:

i) Domestic sewage
ii) Industrial liquid wastes including chemical, paper and food-processing industry wastes
iii) Animal wastes, both solid and liquid
iv) Water course pollution from sewage, industrial wastes and agricultural cropland leachates
v) Highway de-icing compounds
vi) Agricultural chemicals including pesticides and fertilizers
vii) Mining, building and highway constructions
viii) Land disposals of garbage and other domestic wastes through sanitary
ix) Land disposals of industrial wastes through land fills and soil conditioner-compositing procedures

In most of the effluents and wastes, heavy metals are found in an amount sufficient enough to cause toxic effects in plants (Schraufnagel, 1962; Ajmal et al., 1984). Bisessar et al. (1983) found nickel, copper and cobalts upto 15000 ppm 2000 ppm and 66 ppm in concentration respectively in the vicinity of a nickel refinery. Similarly some other workers have also reported soil pollution due

to heavy metals near the industries (Hutchinson and Whitby, 1974; Lagerwerff and Brower, 1974). Gotoh et al.(1979) reported that mercury is also found in amount sufficient to cause pollution naturally in the soil derived from igneous rocks. It has found that mercury is retained in the soil by its colloidal particles (Katsuhiro and Shigenori, 1969). Several workers have reported that mineral mercury compounds, when in excess are transformed into more toxic organic forms like CH_3HgCH_3 and CH_3Hg by the microbial activity of soil. This phenomenon is called as methylation of mercuric compounds (Jenson and Jernelov, 1969; Wiadrowska, 1972; Rogers, 1976; Gotoh et al., 1979). When the trace metals present in rhizosphere are below the required amount, the plants suffer from deficiency. Conversely, when these metals are in excess in soil, the plant suffer from pollution which results in significant losses in yield (Valee and Ulmer, 1972). At present,there is excessive accumulation of heavy metals in soil due to the technological activities of man (Maliszwska et al., 1985). High concentrations of these metals are especially dangerous in arable land. Heavy metals are absorbed by roots and translocated to different parts of the plant body (Haghiri, 1973; Lagerwerff, 1971). The uptake of these soil pollutants is influenced by soil factors like pH, cation exchange capacity, availability of phosphorus etc.

(Miller et al., 1976). Like air pollutants, heavy metals alter the physiology and biochemistry of the plants (Tyler, 1974; Vaituzis et al., 1975).

Several workers have reported adverse effects of industrial effluents, waste materials and heavy metals on the growth and yield of crop plants (Froster, 1954; Hassett et al., 1976; John, 1976; Agarwala et al., 1977; Beckett and Davis, 1978; Heale and Ormord, 1982; Ajmal et al., 1984; Ajmal and Khan, 1984; Hale et al., 1985). Industrial dairy processing effluents have been found to reduce the growth of kidneybean and pearl millet (Ajmal et al., 1984). Ajmal and Khan (1984) irrigated wheat and pea with effluents from Mohan Meakin Breweries Ltd. Ghaziabad, U.P. There was reduction in germination and growth when irrigated with 100% effluents. However, irrigation with 50% effluents was beneficial to plant growth. Goodbold and Huttermann (1985) studied the effect of zinc, cadmium and mercury on root elongation of Picea abies. They observed that mercury was much toxic in inhibiting root elongation in comparision of Zn and Cd. Mercury was about 100 times more toxic than zinc. Similar results were also observed by Graft and Schwantes (1983).

When high levels of heavy metals are found in combinations, the resulting toxic effects on plants appears more severe than the single metal (Hassett et al., 1976). Several

workers have studied interaction of two or more heavy metals on different crop plants (Hutchinson, 1973; Beckett and Davis, 1978). Heale and Ormord (1982) studied the effect of nickel and copper on Acer rubrum, Cornus stolonifera, Lonicera tetrica and Pinus resinosa. The visible symptoms they observed on the plants were development of interveinal leaf chlorosis, interveinal reddening, leaf tip blackening, abnormally small leaves and leaf drop. The symptoms appeared on roots were necrosis, reduction in lateral roots,stunting and thickening. Growth of maple and dogwood was severely retarted at 20 mg/lit. Ni or the combination of 10 mg/lit. Ni and 20 mg/lit Cu. Honeysuckle was much more sensitive to Ni and Cu than maple and dogwood. Needle elongation of pine seedlings was retarted at 20 mg/lit Cu (Heale, 1980). However, there was no distinct pattern of metal effects on pine.

Heale et al. (1985) studied the effect of nickel and copper mixtures on tomato plants. In all cases both Ni and Cu had adverse effects on plant growth whether singly or combined. There was no interactive effect of metals when they were present in combination. However, effects were almost additive without any significance. Nickel was more toxic than copper, as had also been noted earlier (Froster, 1954). Both metals retarded leaf area development of tomato plants with joint action, However, leaf growth was retarded at as low as 0.3 mg/lit Ni and Cu

singly or in combination. However, Sowell et al. (1957) reported threshold for Cu toxicity symptoms in cotton of less than 0.8 mg/lit but greater than 0.4 mg/lit. Patel et al. (1976), however, found that Ni and Cu had a threshold for growth effects on chrysanthemum of greater than 0.06 mg/lit.

Root-knot Nematodes

Root-knot nematodes (Meloidogyne species) are one of the most devastating group of plant parasitic nematodes as they have exceedingly wide host range and are encountered in greatly diverse habitats. Their existence was first recorded by Berkeley (1885) in a greenhouse on cucumbers in England. Since then the pathogens have been known for a considerably long time with different names. The present name Meloidogyne was accorded by Goeldi (1887). Chitwood (1949) re-established the present day genus Meloidogyne describing 4 species and one variety on the basis of characteristics of perineal patterns. Thenafter a number of new species were added to this list. Triantaphyllou (1982) enlisted 57 species of Meloidogyne described so far.

Sasser (1977) gave an account of distribution of root-knot nematode species in different parts of the world. M.incognita (Kofoid and White 1919) Chitwood 1949, M. javanica (Trueb 1885) Chitwood 1949; M.arenaria (Neal 1859)

Chitwood 1949 and M.hapla Chitwood 1949 are the most commonly encountered species; M.incognita is the commonest. Many vegetables suffer considerably due to attack of root-knot nematodes and general loss of the vegetables in different regions of the world due to root-knot nematodes is estimated as 11-25% (Lamberti, 1979; Sasser, 1979).

Root-knot nematodes also have the ability to interact with other plant pathogenic organisms. They interact synergistically with other pathogens and cause greater damage to a number of plants. Most prevalent interactions of root-knot nematodes in nature are with fungi, bacteria and viruses (Table 1).

Interactions of Air Pollutants and Biotic Plant Pathogens

Plant diseases induced by biotic pathogens are influenced by various environmental factors. Air pollution is a relatively new factor that can injure plants and decrease growth and yield (Mudd and Kozlowski, 1975). It can also affect parasitic plant diseases (Heagle 1973 and 1982). There are a number of reports which indicate that incidence of foliar plant diseases caused by fungi are adversely influenced by air pollution. In contrast, enhancement of fungal plant diseases under air pollution stresses are also on record.

Table 1: Interaction of *Meloidogyne* species with other plant pathogens

Meloidogyne spp.	Associated organism	Host	Comments	Reference
M.incognita	*Fusarium oxysporum* f. *nicotianae*, *Alternaria tenius*	Tobacco	Most severe brown spot of leaves in the presence of all three	Powell and Batten, 1969
M.incognita	*Pythium ultimum*, *Culvularia trifolii* *Botrytis cinerea*, *Aspergillus ochraceous*, *Penicillium martensii* *Trichoderma harzianum*	Tobacco	Plant roots predisposed, non-pathogenic fungi caused increased root necrosis	Powell *et al.*, 1971
M.incognita	*F.oxysporium* f. *nicotianae*	Tobacco	Significant infection occurred only when both nematodes and fungus present	Porter and Powell, 1967
M.javanica	*F.oxysporium* f. *nicotianae*	Tobacco		
M.arenaria	*F.oxysporium* f. *nicotianae*	Tobacco		
M.incognita acrita	*Phytophora parasitica* var. *nicotianae*	Tobacco	Severity of blank shank increased	Powell and Nusbaum, 1960
M.incognita	*R.solani*	Tobacco	Worst infection when nematode inoculated 10 or 21 days before *R. solani*	Batten and Powell, 1971

Table 1 (Contd.)

Meloidogyne spp.	Associated organisms	Host	Comments	Reference
M.incognita	*R.solani*	Cotton	Synergism occurred	Carter, 1975
M.incognita acrita	*R.solani*	Cotton	Disease increased	Reynolds and Hanson, 1957
M.incognita acrita	*Verticillium albo-atrum*	Cotton	Severity of wilt increased	Khoury and Alcorn, 1973
M.incognita	*Corynebacterium michiganenis*	Tomato	More bacterial leaf spot	Moura *et al.*, 1975
M.incognita	Secondary invaders	Tomato	Nematode reduced resistance	McClellan and Christie, 1949
Meloidogyne spp.	*F.oxysporum* f. *lycopersici*	Tomato	Nematode reduced resistance	Harrison and Young, 1941
M.incognita acrita	*F.oxysporum* f. *cubense*	Banana	Incubation period of fungus shortened	Loos, 1959
M.javanica	*F.oxysporum* f. *lycopersici*	Tomato	Fungal infection increased	Bergeson *et al.*, 1970
M.javanica	*F.oxysporum* f. *tracheiphilum*	Cowpea	Wilt more severe	Thomason *et al.*, 1959

Table 1 (Contd.)

Meloidogyne spp.	Associated organisms	Host	Comments	Reference
M.javanica	*Macrophomina phaseoli*	Kenaf	Root rot increased	Tu and Cheng, 1971
M.hapla	*Verticillium dahliae*	Strawberry	Control of nematode delayed	Meagher and Jenkins, 1970
M.hapla	*Aspergillus flavus*	Peanut	Greater fungal development	Minton *et al.*, 1969
M.hapla	*F.oxysporum* f. *lycopersici*	Tomato	Susceptibility wilt increased	Jenkins and Coursen, 1957
M.hapla	*Corynebactonium insidiosum*	Alfalfa	Syngestic interaction	Hunt *et al.*, 1971; Norton, 1969; Griffin and Hunt, 1972
M.javanica	*Pseudomonas marginata*	Gladiolus	Scab disease increased	El-Goorani *et al.*, 1974

Interaction of air pollutants with fungal plant pathogens:

Sulphur dioxide:-

SO_2 is reported to influence a number of fungal plant pathogens (Table 2). It has been found that fungal hyphae show resistance to SO_2, especially when protected by the host tissues. SO_2 is used to inhibit post-harvest decay of grapes by Botrytis cinerea, Cladosporium herbarum and species of Alternaria and Stemphylium but concentration of SO_2 required for this effect are more than 200 times those usually found in polluted ambient air (Heagle, 1973). The spores of the most fungi tested have demonstrated great resistance to direct exposure to SO_2, even at high doses (Couey and Uota, 1961; Hibben, 1966). Couey and Uota (1961) reported that only 20% conidia of B.cinerea germinated when exposed to 36 ppm SO_2 for ½ h. However, Hibben (1966) observed no effect on the germination of spores of 10 saprophytic and parasitic fungi exposed on agar to 10 ppm SO_2 for 1-6 h. Moist spores are comparatively more sensitive to SO_2 than dry spores. Couey (1965) obtained 60% reduction in spore germination of Alternaria spp. with treatment of 50 ppm SO_2 for 24 min. but 100 ppm SO_2 was required to produce a similar decrease in the dry spores at 98% R.H.

It has been well recognized on the basis of field observations that obligate fungi are more sensitive to SO_2

Table 2: Interaction of sulphur dioxide (SO_2) and plant pathogenic fungi

Dose	Fungi	Host	Effect	Reference
50 ppm for 2 min at moist condition of 100 ppm for 24 min at 98% RH	Alternaria	-	60% dicrease in spore germination	Couey, 1965
Ambient	Armillaria mellea	Trees	Incidence increased	Donaubauer, 1968; Jancarik, 1961; Kundela and Novakava, 1962
Ambient	Coleosporium sp.	Trees	Disease incidence decreased	Linzon, 1958; Scheffer and Hedgcock, 1955
Ambient	Cronatrium	Trees	Disease incidence decreased	Linzon, 1958; Scheffer and Hedgcock, 1955
0.01 and 0.04 ppm for 2 days after inoculation	Diplocarpon rosae	Rose	Diseased leaflet area slightly increased at 0.01 ppm more than the 0.04 ppm	Saunders, 1966
Ambient	Glocophyllum	Trees	Increased incidence	Jancarik, 1961

Table 2 (Contd.)

Dose	Fungi	Host	Effect	Reference
0.15 ppm for 14 h/d 8 days before inoculation	Helminthosporium maydis	Maize	Decrease in lesion number	Laurence et al., 1979
Ambient	Hypodermella sp.	-	Decrease in disease incidence	Scheffer and Hedgcock, 1955
Ambient	Hysterium pulicare	Alder, birch	Decrease in disease incidence	Skye, 1968
Ambient	Lophodermium piceae	-	Increase in disease incidence	Kudela and Novakova, 1962
Ambient	Melampsora sp.	-	Increase in disease incidence	Scheffer and Hedgcock, 1955
0.30-0.40 ppm for 24-72 h before incubation of conidia	Microsphaera alni	Lilac	Decrease in conidial germination and penetration	Hibbon and Taylor, 1975
90 ppm SO_2 in nutrient solution	Penicillium sp.	-	Slight stimulation in growth	Sounders, 1966
Ambient	Puccinia	Trees	Decrease in disease incidence	Scheffer and Hedgcock, 1955
Ambient	P.graminis	Wheat	Less disease incidence	Johansson, 1954

Table 2 (Contd.)

Dose	Fungi	Host	Effect	Reference
0.10 ppm for 100 h from 2 days for inoculation	P.graminis	Wheat	Decrease in lesion number	Laurence et al., 1979
Ambient	P.striiformis	-	Decrease in germination of uredospores	Sharp, 1967
Ambient	Rhizosphaera kalkhoffii	Red pine	Increase in disease incidence	Chiba and Tanaka, 1968
Ambient	Rhytisma acerinum	Sycamarea	Decrease in disease incidence	Bevan and Geenhelgh, 1976
0.20 ppm for 6 h 5 days after inoculation	Scirrhia acicola	Scots pine	Increase in lesion number	Weidensaul and Darling, 1979
1.0 ppm for 4 h	S.acicola	Agar media	Normal growth	Ham, 1971
Ambient	Trametes serialis and T.heteromopha	Trees	Increase in disease incidence	Jancarik, 1961
0.13 ppm for 24 h/d (on 8 days before or 7 days after inoculation)	Uromyces phaseoli	Bean	Decrease in pustule number, spore size and germination	Weidensaul and Darling, 1979

than other fungi. The rust fungus, Uromyces phaseoli parasitising bean leaves was inhibited by SO_2 (Weinstein et al., 1975). Sulphur dioxide also inhibited Puccinia graminis on wheat when exposed to SO_2 (Laurence et al., 1979). Lilac leaves infected by Microsphaera alni when exposed to 0.40 ppm SO_2 for 24-72 h continuously, there was reduction in spore germination, penetration and hyphal production of the fungus (Hibben and Taylor, 1975). Industrial emmissions containing SO_2 have been found to decrease the incidence of foliar diseases caused by various Ascomycetes including Hypodermella junipera, Rhytisma acerinum, Hysterium pulicare, Venturia inaequalis (Schffer and Hedgcock, 1955; Przybylski, 1967; Skye, 1968). Kock (1935) reported the absence of oak powdery mildew caused by Microsphaera alni near a paper mill. Hibben and Walker (1966) noticed that lilacs grown in polluted air of New York City and other urban areas showed substantially less infection of powdery mildew fungus than lilacs in rural areas. Saunders (1966) exposed roses inoculated with Diplocarpon rosae to low doses of SO_2 for 2 days after inoculation. He found that 0.04 ppm SO_2 considerably decrease the disease while there was slight increase at 0.01 ppm. Saunder (1966) further noted that D.rosae was rarely present on roses in area where daily average SO_2 concentration was greater than 0.04 ppm, but the disease was more frequent in area with less SO_2 concentration.

Laurence et al. (1979) obtained 38% decrease in number of lesions caused by Helminthsporium maydis on maize exposed to 0.15 ppm SO_2 for 14 h daily for 8 days before inoculation. Weinstein et al. (1975) observed that exposures that decrease parasitism of bean by U.phaseoli did not affect parasitism of tomato leaves by Alternaria solani. Scheffer and Hedgcock (1955) recorded decreased parasitism by the species of Cronartium, Coleosporium, Melampsora, Peridermium, Pucciniastrum and Puccinia, where trees were injured by SO_2. With increasing distance from SO_2 source, decrease in plant injury and increase in incidence of fungi were observed.

There are very few reports on the synergistic interaction of SO_2 and fungi on plants. Several workers reported the increase of Armillaria mellea in trees injured by SO_2 (Jancrik, 1961; Scheffer and Hedgcock, 1955; Kudela and Novakova, 1962; Donaubauer, 1968). Similarly, a increased incidence of Lophodermium abietis was found on spruce needles injured by SO_2 by Kudela and Novakova (1962). Weidensaul and Darling (1979) observed that when scot pine seedlings after 5 days of inoculation by Schirrhia acicola were exposed to 0.20 ppm SO_2 for 6 h, exhibited more lesions of the fungi. When seedlings were exposed for 6 h and inoculated after 30 min of exposure, the plants showed similar but non-significant results. Detailed field

studies have shown good negative correlations between the number of Rhytisma acerinum caused spot lesions per unit of leaf area and mean annual concentrations of SO_2 (Bevan and Greenhalgh, 1976).

Fluoride:-

Few attempts have been made to acertain the influence of fluorides on plant pathogenic fungi and viruses (Table 3). Significantly higher levels of fluorides in air inhibit most of the fungi (McCune et al., 1973). Some cultural studies indicate the inhibition of colonial growth of fungi on agar medium containing sodium fluoride. More NaF was required to inhibit Verticillium albo-atrum and Helminthosporium sativum than Pythium debaryanum (Tingey and Blum, 1973). There are few reports on the effect of hydrogen fluoride (HF) on fungi in controlled conditions (McCune et al., 1973; Weinstein et al., 1975). McCune et al. (1973) exposed bean plants to hydrogen fluoride before and after inoculation with U.phaseoli. Inhibition in both in number and growth of uredia in leaves was observed and F levels were increased to 600-1300 ppm. In second experiment, pustule numbers were reduced by HF exposure only before inoculation while in third experiment both pre-and post inoculations caused inhibition of the rust (McCune et al., 1973). The effects of HF on parasitism of tomato by Alternaria solani and Phytophthora infestans were also

Table 3: Interaction of fluorides and plant pathogenic fungi and viruses.

Treatment	Fungus/Viruses	Host	Effect	Reference
HF, before inoculation	*Alternaria solani*	Tomato	Development of fungus inhibited when HF level in leaves were 200-500 ppm	McCune *et al.*, 1973
NaF in high dose	*Helminthosporium sativum*	Media	Inhibition of colony growth	Heagle, 1973
HF in high dose pre- and post-inoculations	*Uromyces phaseoli*	Bean plants	Inhibition in number and growth of uredia	McCune *et al.*, 1973
H,F post-inoculation	*Phytophthora infestans*	Tomato	HF inhibited fungal growth	McCune *et al.*, 1973
100-300 ppm Fluoride	T M V	Bean leaves	Number of TMV lesions increased	Treshow *et al.*, 1967
H F	T M V	Bean leaves	Reduction in TMV particles and infection when leaves contain more than 500 ppm	Dean and Treshow, 1965

studied by McCune et al. (1973). Pre-inoculation exposures of tomato plants to HF caused decreased development of A.solani, mainly on young leaves where HF levels were 200-500 ppm. The post-inoculation exposures had little effect on the fungal development. Pre-inoculation exposures did not change disease development caused by P.infestans, where as the effects of post-inoculation exposure were inconsistent.

Ozone (O_3):-

Ozone while influencing a number of parasitic fungi inhibits or enhances the parasites (Heagle, 1973) (Table 4).

Ozone is more effective than SO_2 in decreasing spore germination. The degree of sensitivity of O_3 depends upon the doses, fungus.species, spore morphology, moisture and substrate. Multi-celled pigmented spores and spores with thick cell walls, are usually more resistant to O_3 than single-celled spores or those with hyaline or thin cell walls. For example, percentage germination of the large multi-celled conidia of Macrosporium sp. was greater than that of smaller, single-celled spores of Sclerotinia fructicola and Penicillium expansum at 0.60 ppm O_3 for 1-3 h on freshly cut apples (Smock and Watson, 1941). Unlike the SO_2, dry spores are more sensitive to ozone than wet spores (Heagle, 1973).

Table 4: Interaction of ozone (O_3) and plant pathogenic fungi

Treatment	Fungi	Host	Effect	Reference
Ambient	Botrytis cinerea	Potato	Severe infection on O_3 injured leaves of potato	Manning et al., 1969
Very low doses	B.gladiolorum	Gladiolus flower	Inhibit development of disease	Magdycz and Manning, 1973; Magie, 1963
-	Botrytis spp.	Chrysanthemum	Decreased disease in petals	Magie, 1963
0.07-0.10 ppm 10 h/day for 15 days before inoculation	B.cinerea	Potato	90-100% leaf showed symptoms of fungal disease	Manning et al., 1970
Large doses	B.cinerea	Straw berries	Invasion not decreased, growth of aerial mycelium suppressed	Spalding, 1966
-	Fusarium oxysporium	Plant	20% more fungal colonies on root of plant with leaves injured by O_3	Manning et al., 1971b
0.10 ppm 8 h/day for 10 weeks	Fusarium f. conglutinans	Cabbage seedlings	No significant effect	Manning et al., 1971a
0.06 and 0.12 ppm 6 h/day for 6 days before inoculation	Helminthosporium maydis	Maize	Sporulation increased	Heagle, 1973

Table 4 (Contd.)

Treatment	Fungi	Host	Effect	Reference
Ambient	Heterobasidion annosum	Pine trees	O_3 injured needles had more fungal infection	James et al., 1980
0.50 ppm continuously	Monilinia fructicola	Peaches	Decreased fungal disease	Spalding, 1966
0.10 ppm 6 h, 10 days of inoculation	Puccinia coronata	Oat	Uredia became significantly smaller	Heagle, 1970
0.06, 0.12, 0.18 ppm 8 h/day	P.graminis	-	Decreased sporulation and growth of hyphae	Heagle and Key, 1973
1 ppm for 15 days	P.expansum	Oranges	Less sporulation, invasion and decay	Harding, 1968
1 ppm for 15 days	P.digitatum	Lemons	Less invasion, sporulation and decay	Harding, 1968
0.25 ppm - 6 h	Rhizopus stolonifer	-	Spore germination was inhibited	Hibben and Stotzky, 1969
0.10 ppm-14 h/day one day after inoculation	Uromyces phaseoli	Beans	Uredia became significanly smaller in size and plants became resistant to fungus	Heagle, 1973

There are considerably more reports and sound evidences that O_3 inhibits the fungal infection. Magie (1963) and Manning (1971) observed inhibition of Botrytis gladiolorum on gladiolus flower when the plants were exposed to O_3 at a dose too low to cause injury to gladiolus. Similarly, in the case of chrysanthemum petals, ozone decreased disease by Botrytis sp. even though the petals were injured by O_3 (Manning, 1971). Heagle (1970) exposed 10 varieties of oat infected with Puccinia coronata at 0.10 ppm O_3 for 6 h after 10 days of inoculation. Slight injuries in all plants due to O_3 and significant reduction in size of uredia occurred. Heagle and Key (1973) observed inhibition in sporulation of P.graminis when infected wheat plants were exposed to low doses of O_3 (0.06, 0.12, 0.18 ppm for 6 h/day). The decreased sporulation appeared to be related to O_3 injury to host mesophyll cell and decreased growth of hypahe. Heagle and Strickland (1972) reported 84%, 84% and 75% inhibition in penetration of Erysiphe graminis in barley leaves when exposed to 0.05, 0.10 and 0.15 ppm O_3 respectively. Similar effects were observed when barley leaves having mature canidia exposed to 0.10, 0.20 and 0.30 ppm O_3 for 24 h during incubation. However, Schuette (1971) observed that under high doses of O_3, the mature spores remained unaffected.

There are substantial evidences which support ozone injury can predispose plants to disease caused by

facultative parasites (Manning et al., 1969). Manning et al. (1970) were first to report synergistic interaction between O_3 and Botrytis cinerea on potato. Infection and invasion of potato leaves by B.cinerea were greatly increase when leaves were injured by O_3 (0.15 - 0.25 ppm 6-8 h) before inoculation. In one Geranium cultivar 90-100% of the leaf area showed symptoms of disease caused by B.cinerea when leaves were injured by O_3 at 0.07-0.10 ppm concentration for 10 h/day for 15 days, before inoculation. Similarly broad bean plants exposed to 0.15 ppm O_3 for 8 h before inoculation of B.cinerea, showed significantly increased infection and invasion of the fungus (Magdycz, 1972). Wukasch and Hofstra (1977) obtained similar effect of O_3 when onion plants injured by O_3 in open top field chamber before were inoculated with B.squamosa. The infection was 2 times more. Further, they showed significantly less infection of B.squamosa by spraying the plants with an antioxidant chemical (DDX-4891). This supported that O_3 injury predisposed plants to Botrytis sp. The reaction of O_3 on H.maydis infecting maize leaves was negative or positive, depending upon the time of exposure in relation to stage of fungus development as well as on the concentration of O_3 (Heagle, 1977). When maize plants exposed to O_3 for 6 h per day before 6 days of inoculation, sporulation increased at 0.06 ppm and 0.12 ppm but not at 0.18 ppm. When plants were exposed after 6 days of inoculation

there was decrease in sporulation with increase in O_3 concentration.

There are a few reports onthe interaction of O_3 and fungus parasites of root. James et al. (1980) studied the combined effect of O_3 and Heterobasidion annosum on pine trees. Trees, with different degree of sensitivity of O_3, were inoculated with H.annosum in an O_3 polluted area near Los Angeles. One year later, infection and colonization of H.annosum on the needles and their premature fall due to O_3 injury were recorded, while the healthy needles had no infection.

A persual of the literature indicates that O_3 alters the parasitism primarily through effect on host plant. Facultative parasites are affected more than facultative saprophytes such as H.maydis and Cercospora arachidicola (Heagle, 1973).

Acid rain :-

According to Bruck and Shafer (1984) aic precipitation represents an alternation in the environment component. Hypothetically, it could have three effects on a disease situation:

i) Within the range of acidity currently occuring in precipitation no change may be evident or the change is simply not sufficient to alter the otherwise predictable course of the host-parasite interaction.

ii) Acid rain may influence host resistance, pathogen virulence or pathogen inoculum density in such a way as to increase disease incidence or severity on a plant or within a plant population

iii) Acid rain may decrease plant susceptibility, parasite virulence or parasite inoculum to such an extent that the disease process is decreased in severity or indicence

The pH has great influence on the growth and reproduction of many fungi, directly or indirectly (Mehrotra, 1980). Effect of simulated acid rain has been studied on a few fungal pathogens (Table 5). Simulated acid rain caused inhibition of fusiform rust (Cronartium fusiforme) on leaves of yellow oat inoculated with aeciospores. The number of infections and telia were decreased when sulphuric acid of pH 3.2 was applied on each of 14 days before and after inoculation (0.63 Cm rainfall applied over 10 min per day) (Heagle, 1982). The number of H.maydis (Race T) lesions on the maize were increased when conidia were incubated in water at pH 3.5 and leaves were subsequently treated with "rain" at pH 3.5 (0.63 cm over 10 min/day for 21 days). Shriner (1978) treated kidney bean plants infected by Uromyces phaseoli and Meloidogyne hapla, three times weekly with simulated rain at pH 6.0 or 3.2. 'Rain' at pH 3.2 caused temporary inhibition of rust severity

Table 5: Interaction of acid rain with plant pathogens

Treatment	Pathogen	Host	Effect	Reference
H_2SO_4 of pH 3.2 14 days pre-and post-inoculation	Cornartium fusiforme	Yellow oat	Number of telia and infection decreased	Heagle, 1982
Simulated acid rain at pH 3.2 (0.63 cm over 2 min/day for 21 days)	Uromyces phaseoli + Meloidogyne hapla	Kidney beans	Fungal and nematode diseases decreased	Shriner, 1978
Simulated acid rain at pH 3.2 (0.63 cm over 10 min/day for 10 days)	Pseudomonas phaseolicola	Kidney beans	Increased bacterial disease	Shriner, 1978

and decreased root infection and reproduction of root-knot nematode.

Interaction of air pollutants and plant pathogenic bacteria and nodule forming bacteria :

Sulphur dioxide :-

SO_2 is also known to influence bacterial plant pathogens (Table 6). Recently, Laurence and Aluiso (1981) reported the effects of SO_2 on the parasitism of bacteria. SO_2 was found to reduce the rate of lesion development and maximum lesion size, when SO_2 exposures were sufficient to increase sulphur contents of leaves. In some cases exposure duration in relation to inoculation play important role. Corynebacterium nebraskense was inhibited when maize plants were exposed continuously to 0.20 ppm SO_2 for 24 h daily, 5 day before inoculation, 2 days after inoculation and both pre-and post inoculation. However, maximum inhibition of bacteria occured at 2 day post-inoculation exposure. In the same study, Laurence and Aluiso (1981) also studied SO_2 effect on Xanthomonas phaseoli. Inhibition in X.phaseoli occurred when soybean plants were exposed to 0-10 ppm SO_2 for 24 h 5 days before inoculation, 5 days after inoculation and both 5 days before and after inoculation. Inhibition of bacteria was maximum at pre-and post-inoculation exposures.

Table 6: Interaction of sulphur dioxide(SO_2) and plant pathogenic bacteria and viruses

Treatment	Bacteria/viruses	Host	Effect	Reference
0.20 ppm for 24 h daily 5 days before inoculation 2 days after inoculation or both	Corynebacterium nebraskense	Maize	Inhibitory	Laurence and Aluisio, 1981
0.10 ppm for 24 h daily for 5 days before inoculation 5 days after inoculation or both	Xanthomonas phaseoli	Soybean	Inhibitory effect	Laurence and Aluisio, 1981
0.10 ppm for 24 h for 7 days	Southern bean mosaic virus	Bean	Increase in virus concentration	Laurence et al., 1981
0.10 ppm for 24 h for 7 days	Maize dwarf mosaic virus	Maize	Increase in virus concentration, infection and severity of symptoms	Laurence et al., 1981

Ozone (O_3):-

Ozone is reported to inhibit plant pathogenic bacteria (Table 7). Laurence and Wood (1978) observed decreased number of lesions of Pseudomonas glycinea on soybean leaves at 0.08 or 0.25 ppm O_3 for 4 h from 16 days to 1 h before inoculation or 1 h to 1 day after inoculation, but not after 2 days of inoculation. Similarly, O_3 at 0.20 ppm for 4 h decreased the number of lesions caused by Xanthomonas fragariae on wild strawberry.

Some researches indicate that bacteria protect leaves from O_3 injury but other reports indicate the lack of a protective effect (Heagle, 1982). Temple and Bisessar (1979) observed that Xanthomonas phaseoli provides some protection to white bean leaves from ambient oxidants in Canada, but the effect was not commercially significant. However, in controlled conditions, X.phaseoli did not provide such protection to navy bean from 0.24 ppm O_3 over 8 h for 1,2 or 4 days after inoculation (Olson and Saettler, 1979). Pratt and Krupa (1979) noticed a generalized protection of soybean by P.glycinea when exposed to O_3 for 1-3 days after inoculation. Pell et al.(1977) reported that acute foliar injury caused by Pseudomonas sp. would protect soybean leaves from acute injury caused by O_3 and vice-versa. Howell and Graham (1977) also observed antagonistic interaction between O_3 and X.alfalfae on alfalfa plants.

Table 7: Interaction of ozone (O_3) and plant pathogenic bacteria

Treatment	Bacteria	Host	Effect	Reference
0.08 or 0.25 ppm 4 h, from 1 h-16 h days before inoculation	Pseudomonas glycinea	Soybean leaves	Decreased number of lesions	Laurence and Wood, 1978
1-3 days after inoculation	P.glycinea	Soybean	Severe bacterial infection protected soybean from acute O_3 injury, antagonistic effect	Pratt and Krupa, 1979
High dose	P.glycinea	Soybean	Severe bacterial infection protected soybean from acute O_3 injury, antagonistic effect	Pell et al., 1977
0.20 ppm - 4 h	Xanthomonas fragariae	Strawberry	Decreased number of lesions	Laurence and Wood, 1978
Ambient	X.phaseoli	Bean leaves	Antagonistic effect	Temple and Bisessar, 1979
0.24 ppm - 8 h; 1,2, 3 days after inoculation	X.phaseoli	Navy beans	No effect	Olson and Saettler, 1979

They suggested that disease on alfalfa caused by X.alfalfae was decreased due to O_3 exposure and that bacterial infection reduced O_3 injury.

Effect of ozone on nodule-forming bacteria have also been demonstrated. Reinert et al. (1971) reported decreased number of Rhizobium nodules on soybean when plants were exposed to 0.12 - 0.15 ppm O_3 for 15 days. However, Manning et al. (1972) observed reduction in number, size and weight of Rhizobium nodules on pinto bean roots at 0.06 ppm O_3 for 8 h per day for 20-50 days. Tingey and Blum (1973) found similar results when 3-week-old soybean plants were exposed to 0.75 ppm O_3 for 1 h. O_3 decreased the leghaemoglobin contents in plant root. It is speculated that effect of ozone is direct on bacterial cells because it is highly reactive and probably breaks-down in the soil, or O_3 affects plant foliage resulting in alteration in root physiology. Another possibility is the change in quality or quantity of root exudates. Increased rate of lignification of root cells have also been advocated as possible reason for inhibition of nodulation (Scott and Lesher, 1963).

Other air pollutants:-

Fluoride may decrease the disease caused by P. phaseolicola (Heagle, 1973). However, Heagle (1982)

noticed that disease caused by P.phaseolicola in bean leaves was not affected by HF exposure at pre-or post-inoculation even at the concentration where 700 ppm fluoride accumulated in the leaves.

Some results indicate that acid rain at levels that can occur in the field might significantly affect parasitism (Heagle, 1982). Shriner (1978) observed increased disease symptoms in kidney beans by Pseudomonas phaseolicola when plants were stressed by simulated acid rain at pH 3.2 before inoculation (0.63 cm over 10 min per day for 10 days). Shriner (1978) also observed decreased incidence of Rhizobium nodulation in red kidney beans and soybean in glasshouse and kidney beans in field when stressed to acid rain of pH 3.2. However, Feicht (1981) reported no effect of biweekly acid rain at pH 2.8 on Rhizobium nodulation in soybean.

Manning (1971) suggested that limestone dust deposits on plant leaves can affect bacterial populations. He isolated more colonies of unidentified bacteria from grape and sassafras leaves covered with limestone dust.

Interaction of air pollutants and plant viruses:

Sulphur dioxide :-

A few plant viruses are known to be affected by SO_2 (Table 6). SO_2 has been found to increase viruses in bean

and maize (Laurence et al., 1981). An increase in southern bean mosaic virus and sulphur contents in bean leaves were noted when plants were exposed to 0.10 ppm SO_2 for 7 days continuously with pre- and post-inoculations. However, in maize plants, increase of maize dwarf mosaic virus (MDMV) leading to greater infection and more severe symptoms were observed without any increase in sulphur contents of the leaf, when plants were exposed continuously to SO_2 pre-or post-inoculations of plants.

Fluoride :-

Fluoride is known for its predisposing effects on pinto bean leaves to TMV infection. The number of TMV lesions was greater on inoculated bean leaves than on the controls when leaves contained 100-300 ppm fluoride (Dean and Treshow 1965; Treshow et al., 1967). If leaf tissue contained fluoride more than 500 ppm, reduction in TMV particles and infection was observed by Dean and Treshow (1965).

Ozone :-

Synergistic or antagonistic interactions between ozone and plant viruses have been reported (Table 8). Bernnen and Leone (1970) observed more local lesions on pinto bean leaves inoculated with TMV when plants exposed to 0.10 ppm O_3 for 3 h, 3 or 24 h after inoculation.

Table 8: Interaction of ozone (O_3) and plant viruses

Treatment	Virus	Host	Effect	Reference
0.25 ppm-4 h, 4,5 6 days after inoculation	Bean common mosaic virus	Pinto bean	Less infection	Davis and Smith, 1974b
0.10 ppm - 3 h 3 or 24 h after inoculation	TMV	Pinto bean leaves	More local lesions	Bernnen and Leone, 1970
Ambient	TMV	Tobacco	TMV reduced O_3 injury by 60%	Bisessar and Temple, 1977
0.25 ppm- 4 h, 9 days after inoculation	Tobacco etch virus	Tobacco	Tobacco etch virus protected tobacco leaves from O_3	Moyer and Smith, 1975
0.30 ppm- 3 h for 1 or 2 days after 3 weeks of inoculation	Tobacco streak virus	Tobacco	Significantly more O_3 injury	Reinert and Gooding, 1978
0.40 ppm- 4 h, 6,8, 10 days after inoculation	Tobacco ring	Soybean	TRSV reduced O_3 injury of soybean	Vargo et al., 1978

The effect was, however, absent when exposure occurred 0 or 48 h after inoculation.

There are several reports about the antagonistic interaction of viruses and O_3. Davis and Smith (1974b) noticed partial protection of pinto bean leaves from O_3 (0.25 ppm for 4 h) when inoculated with bean common mosaic viruses 4,5 or 6 days before exposure. Several other viruses (tobacco ring spot, tomato ring spot, alfalfa mosaic and tobacco mosaic) also provided some protection from O_3 injury to primary leaves of pinto beans when plants were inoculated 5 days before exposure (0.25 ppm for 4 h) (Davis and Smith, 1974a). Protection was confined to the area around the lesions for all viruses except tobacco ring spot virus which provided a more general protection. Bisesser and Temple (1977) observed 60% less injury due to O_3 in field grown tobacco infected by TMV than non-infected virus field. Tobacco etch virus protected tobacco leaves from O_3 (0.25 ppm for 4 h) when plants were exposed 9 days after inoculation (Moyar and Smith, 1975). Vargo et al. (1978) exposed soybean plants to 0.40 ppm O_3 4 h after, 6,8,12 days of inoculation with tobacco ring spot virus. They observed less O_3 injury in inoculated plants. However, Reinert and Gooding (1978) reported that systemic infection by tobacco streak virus caused tobacco plants to suffer significantly more injury of O_3 than non inoculated one

when exposed to 0.30 ppm O_3 for 3 h on 1 or 2 days at 3 weeks after inoculation.

The mechanism for virus induced changes in plant response to O_3 are not known. Decreased stomatal conductance has been suggested as a mechanism for protection, but this was not true for tobacco ringspot virus in soybean (Vargo et al., 1978) or for TMV in tobacco or pinto bean (Brennan, 1975).

Interaction of air pollutants and plant parasitic nematodes:

There are a very few reports on the effect of air pollutants and host-parasite interaction involving plant nematodes (Table 9). The existing literature on this topic is mainly concerned with sulphur dioxide and ozone. Bassus (1968) reported increased populations of saprophagous and predaceous nematodes in areas of forest severly damaged by SO_2 and alkaline particulate material than those slightly damaged forest areas. Kozlowska (1981) observed harmful effect of industrial dust on the nematode Panagrolaimus rigidus. Weber et al. (1979) studied the effect of O_3 and SO_2 on reproduction of four plant parasitic nematodes and their host-parasite relationships. They exposed soybean plants to O_3 and SO_2 singly and in combination, and inoculated with Belonolaimus longicaudatus, Heterodera glycines, Paratrichodorus minor and Paratylenchus penetrans. Exposure

Table 9: Interaction of air pollutants and plant parasitic nematodes

Treatment	Nematodes	Host	Effect	Reference
Industrial dust	Panagrolaimus rigidus	-	Harmful effect on nematode	Kozlowska,1978
O_3 and O_3+SO_2	Heterodera glycines	Soybean	Inhibition in reproduction and development of nematode	Weber et al.,1979
O_3 and O_3+SO_2	Belonolaimus longicaudatus	Soybean	Unaffected	Weber et al.,1979
SO_2	Paratylenchus penetrans	Soybean	Enhanced reproduction of nematode	Weber et al.,1979
O_3 and O_3+SO_2	Paratrichodorus minor	Soybean	Reproduction and development of nematode inhibited	Weber et al.,1979
O_3 and O_3+SO_2	Aphelenchoides fragariae	Begonia	Decreased foliar injury by nematode	Weber et al.,1979
O_3 and O_3+SO_2	H.glycines + Rhizobium	Soybean	Severe inhibition in nodules	Weber et al.,1979
	B.longicaudatus+ Rhizobium	Soybean	Inhibition in number of nodules	Weber et al.,1979
	P.minor+Rhizobium	Soybean		

Table 9 (Contd.)

Treatment	Nematodes	Host	Effect	Reference
0.2 ug O_3/lit 0.2 ug SO_2/lit singly or in combination	P.penetrans	Tomato cv. "Walter"	Synergistic interaction	Shew et al., 1982
0.2 ug O_3/lit + 0.8 ug SO_2/lit	P.penetrans	Tomato cv. "Walter"	Antagonistic interaction	Shew et al., 1982
80 ppm O_3(Ambient)	Meloidogyne hapla	Tobacco cv. "virginia-115"	Plants with nematodes suffered more O_3 injury	Bisessar and Plamer, 1984
Simulated acid rain of pH 6.0 or 3.2, 3 times weekly	M.hapla	Kidney beans	Decreased nematode infection at pH 3.2	Shriner, 1978

of infected soybean plants to O_3 and O_3-SO_2 mixture inhibited reproduction and development of H.glycines and P.minor but infection of B.longicandatus was unaffected. Soybean plants exposed to SO_2 showed enhanced reproduction of P.penetrans compared with that of plants exposed to the charcoal filtered air or to O_3. When begonia plants were exposed to pollutants singly or in combination and inoculated with Aphelenchoides fragariae (a foliar nematode) foliar injury of begonia by O_3 and O_3-SO_2 inhibited the nematode. The suppressive effect on A.fragariae was greater in leaves pre-exposed to O_3 or O_3-SO_2 mixture before, rather than after nematode inoculation. The growth of nematode infested soybean plants and leaves of begonia was inhibited by O_3 and the O_3-SO_2 mixture compared with control inoculated with nematode in charcoal filtered air. Nodulation in soybean plants inoculated with B.longicaudatus and P.minor was also suppressed by O_3 and O_3-SO_2 mixture. The inhibition of nodulation in soybean by H.glycines was extensive.

Shew et al. (1982) conducted an experiment to understand interaction of P.penetrans and O_3 and SO_2 on tomato. They exposed tomato cv. "Walter" plant inoculated and uninoculated with 400 larvae of P.penetrans to 0.4 µl O_3/l of air, 0.2 µl O_3/l, 0.2 µl SO_2/l, 0.8 µl SO_2/l singly or in combination for 3 h. They observed synergistic interaction at 0.2 µl O_3/l, 0.2 µl SO_2/l singly and in combination.

However, a mixture of 0.2 ul O_3 and 0.8 ul SO_2 per liter of air showed antagonistic reaction. Bisesser and Palmer (1984) worked out the effect of O_3 on Meloidogyne hapla. They transplanted seedlings of tobacco cv. Virginia-115 inoculated and uninoculated with root-knot nematode larvae (M.hapla) in the field, where O_3 concentration was 80 ppb. Some seedlings were also sprayed with an anti-oxidant, EDU (aethylenediurea). Ambient O_3 inhibited growth and yield of tobacco regardless of inoculation or non-inoculation with M.hapla. Tobacco inoculated with the nematode,however, suffered more ozone injury than uninoculated. There was 20% less galling in inoculated plants sprayed with EDU than non-sprayed. Shriner (1978) reported decreased root infection and reproduction of M.hapla on kidney beans when M.hapla and Uromyces phaseoli infected plants were stressed with simulated rain at pH 3.2 three times weekly.

Interaction of Soil Pollutants and Biotic Plant Pathogens

Interactions between biotic pathogens and soil pollutants, especially heavy metals have received considerably less attention. The use of waste water for irrigating crops may raise serious problems for plants. Pollutants of these kinds are known to enchance the prevelance of root diseases and predispose the plants to pathogenic damage (Cole et al., 1969). The prevelance of root disease may increase due to presence of several plant pathogenic fungi

in sewage water. Cooke (1956) isolated species of Alternaria, Aspergillus, Cephalosporium, Chaetomium, Cladosporium, Coniothyrium, Curvularia, Fusarium, Gliocladium, Mucor, Pencillium, Rhizopus, Scopulariopsis, Stemphyllium and Trichoderma from sewage and polluted water. He also found that various potential plant pathogens reproduce well in sewage and sewage-treatment facilities. Several other investigators have also reported the presence of several plant pathogenic fungi in sewage and polluted water (Cooke, 1954 and 1957; Becker and Shaw, 1955; Cooke and Hirsch, 1958).

It has now been well recognized that heavy metals affect adversely the microflora of soil. Waste-waters and industrial effluents are known to contain various heavy metals. The disturbance to the biological equlibirium in such soils due to the excess of heavy metals may have an unfavourable influence on soil fertility, plant development and yield (Van Facessen, 1973; Kobus and Kabata-Pendias, 1977; Maliszewska et al., 1979; Bisessar, 1982). Bisessar (1982) collected soil from several sites near lead smelter plant. He found higher concentrations of lead,arsenic, cadmium and copper and low pollutation counts of bacteria, actinomycetes, fungi, nematodes etc. close to the smelter. He concluded that low populations of microbes may be because of the toxic effects of heavy metals present in

the vicinity in higher concentrations. Maliszewska et al. (1985) studied effect of heavy metals on micro-organisms by inoculating solutions of Hg, Pb, As, Zn and Cu artificially in plastic pots. They observed the harmful effects of these metals and toxicity was in this order i.e. Hg > As > Zn > Pb. They further pointed out that out of the micro-organisms studied the fungi and actinomycetes were more resistant to heavy metals.

A persual of literature shows that there is voluminous work on effects of heavy metals on plants and soil micro-organisms. The effect of heavy metal pollution of soil on prevelence of root diseases and predisposition of plants has received not much attention. Taking an initiative in this direction, Bisessar et al. (1983) worked out the effects of heavy metals on the celery plants when infected with biotic pathogen (root-knot nematode, Meloidogyne hapla). They transplanted inoculated and uninoculated celery plants with 1000 second stage larvae of M.hapla, in the vicinity of Nickel Refinery where concentrations of Ni, Cu and Co were upto 15000 ppm, 2000 ppm and 66 ppm respectively. Celery plants suffered with severe galling on root in metal contaminated soil. The interactive effect of heavy metals and M.hapla was synergistic. Heavy metal deposition in celery plants was maximum in root and minimum in stalk. Overall deposition of heavy metals was significantly higher

in the plants infected with M.hapla than in non-infected, but grown in same metal contaminated soil. Recently Khan et al. (1986) reported significant reduction in penetration of M.incognita in pepper roots grown in sand with 100-8000 ppm concentrations of cobalt. They concluded that inhibition in penetration may be because of direct toxic effect of cobalt on the nematode.

A perusal of literature on environmental pollution and plant effects in closer perspective and critical analysis of informations available leads one to conclude that environmental pollution affects plant life directly and its impact is tremendous on its various life processes leading to manifestation of a variety of abnormalities depending upon the type of the plant, kind of pollutant and other related factors. Biotic pathogens thriving on plants also influenced directly or their host-parasite relationships are affected and altered. Such pollution induced imbalances may be detrimental. In view of alarming environmental pollution as world-wide phenomenon, consequences on plant diseases caused by biotic pathogens requires substantial study before several basic questions related to the problem can be satisfactorily answered. The studies carried out so far are too meager and are largely restricted to fungal plant diseases. A few initiatives have been taken with bacterial plant pathogens and some plant viruses. Results of some studies show a definite trend in influences of environmental pollution

but in some instances these are contrasting and conflicting. Plant nematodes though a major group of plant pathogens have received negligible study in relation to environmental pollution. The situation dictates for thorough study of the problem involving various nematode species and pollutants in different crop pathosystems. Perhaps, pursuance of such study is one of the most urgent needs of the modern Plant Pathology.

MATERIALS AND METHODS

Collection of Meteorological Data

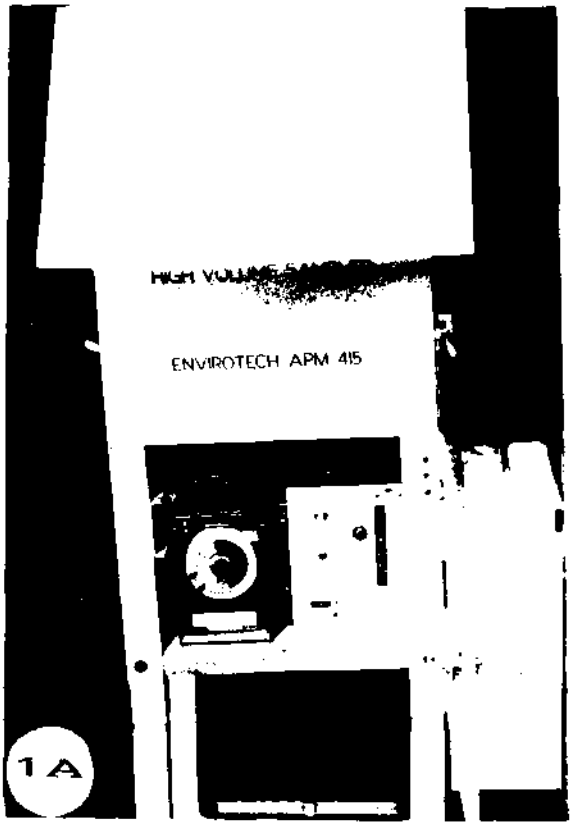
The meteorological data of the area where the Thermal Power Plant, Kasimpur is situated in relation to wind velocity, temperature, relative humidity, rainfall etc. will be collected from the Meteorological Department (Government of India), Aligarh unit.

Collection of Base-line Data on Air Pollutants

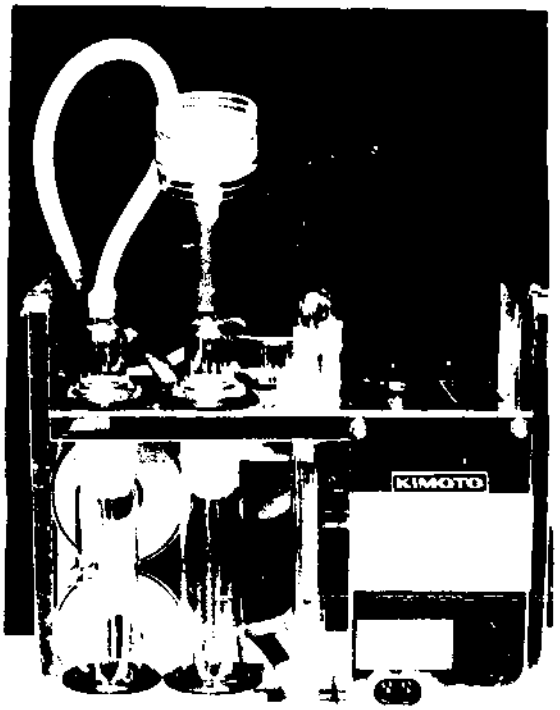
Base-line data on different air pollutants present in the ambient air around the Thermal Power Plant, Kasimpur will be collected. The concentrations of various air pollutants in different seasons (summer, rainy and winter) and at different distances from the source of origination will be determined. For the purpose, air samples will be collected by Handy Air Sampler and High Volume Air Sampler (Figs.A1 and A2) at appropriate times during the seasons from different distances and samples will be analysed (Anon.,1986).

Sampling

For gaseous air pollutants, sampling will be done either by High Volume Air Sampler (Envirotech, New Delhi)



High Volume Air Sampler



Handy Air Sampler

Dynamic State Exposure Chambers

or by Handy Air Sampler (Kimoto, Japan) at the rate of 200 ml to 2 lit of air per minute for 30 min to 24 h.

Methods being followed at the National Environmental Engineering Research Institute (NEERI), Nagpur,(Anon.,1986) will be employed for sampling of various gaseous and particulate air pollutants. Sampling and analysis will be done for SO_2, NO_2, NH_3 and O_3 in gaseous air pollutants and for particulate matter. The obsorbing media to be used for various gaseous air pollutants are as follows:

S.No.	Gaseous air pollutant	Absorbing media for sampling
1.	SO_2	Sodium tetra chloromercurate solution
2.	NO_2	Sodium hydroxide sodium arsenite solution
3.	NH_3	Diluted sulphuric acid
4.	O_3	Alkaline potassium iodine solution

Sampling of ozone (O_3):-

The O_3 will be sampled by either High Volume Air Sampler or Handy Air Sampler at the rate of 1/2 cubic meter per minute for 1/2 - 4 h. Alkaline KI solution will be used as absorbing medium. To avoid the SO_2 interference, Cr_2O_3 impregnated paper will be used. The

absorbing medium and Cr_2O_3 paper will be kept in the impinger as follows-

(1) ———— (2) ———— (3) ————

Midget impinger containing Cr_2O_3 paper	Absorbing impinger	Trap	Flow regulator

Preparation of Cr_2O_3 paper

2-5 g of Cr_2O_3 will be mixed in a petridish with 15 ml distilled water and 0.7 ml of conc. H_2SO_4. The mixture will be pasted over 60 square inch of ordinary filter paper and will be dried at 80-90°C for 1 h in hot air oven. Then the paper will be cut in strips without touching by hand.

Analysis of sulphur dioxide :-

Analysis of SO_2 in samples will be done by Weast and Fack Method (being used at NEERI). Some details of the analysis, preparation of chemicals are described stepwise, below:

(i) **Absorbing reagent (Sodium tetrachloromercurate)**

27.2 g mercuric chloride and 11.7 g sodium chloride will be dissolved in 1 lit double distilled water (D.D.W.) This solution can be stored at room temperature and can be used for two months.

ii) **Sulphamic acid** (0.6%)

0.6 g sulphamic acid will be dissolved in D.D.W. to bring the volume to 100 ml.

iii) **Formaldehyde solution** (0.2% to be prepared fresh)

0.5 ml formaldehyde solution (40%) will be diluted in D.D.W. upto 100 ml.

iv) **Rosaniline hydrochloride solution** (0.2%)

a. **Step I solution**

0.2 g rosaniline hydrochloride will be dissolved in 1 N hydrochloric acid to bring the volume upto 100 ml. It will be kept in refrigerator over night.

b. **Step II solution**

4 ml of step I and 6 ml concentrated HCl will be dissolved in D.D.W. to bring the volume to 100 ml.

v) **Potassium dichromate solution** (0.1N)

Exactly 1.226 g dried potassium dichromate will be dissolved in D.D.W. to 250 ml.

vi) **Sodium thiosulphate** (0.1N)

7.0 g sodium thiosulphate will be dissolved in 250 ml D.D.W. For preservation 2 ml of chloroform will also be added.

vii) **Iodine solution** (0.1N)

10 g of potassium iodine will be dissolved in 20 ml D.D.W. After adding 3 g crystals of iodine, solution will be stirred. Solution will be kept over-night and diluted upto 250 ml with D.D.W. Solution will be preserved in amber coloured bottle in dark.

viii) **Potassium iodine solution** (100%)

10 g of potassium iodine will be dissolved in 10 ml D.D.W. This may be prepared at the time of titration.

ix) **Starch solution**

A paste of 1.25 g soluble starch will be prepared with D.D.W. and will be poured in 250 ml boiling D.D.W. It will be boiled for 5 minutes with stirring and allowed to cool and stand. Fresh and clear supernatent will be used.

x) **Metabisulphite solution**

0.3 g sodium metabisulphite will be dissolved in 500 ml D.D.W. This solution will contain 320 or 400 ug/ml of SO_2.

Standarisation of sodium thiosulphate

50 ml D.D.W. will be taken in 250 ml conical flask and 10 ml potassium dichromate (0.1N), 10 ml H_2SO_4 (1:1) and 1 ml of potassium iodine solutions will be added. The flask will be kept in dark for 5 min for reaction.

Above solution will be titrated with sodium thiosulphate (vi) till a faint yellow colour is obtained. Then 1 ml of starch solution will be added; blue colour will now appear. The titration will be continued until faint blue colour disappears. Final colour will be a distinct bluish green tinge due to chromous ions. Titration reading will be noted. This will be repeated to get constant reading. Then normality of sodium thiosulphate will be calculated according to the following formula:

$$\text{Normality of thiosulphate} = \frac{\text{Normality of dichromate} \times \text{Vol. of dichromate}}{\text{Vol. of thiosulphate required}}$$

Standardisation of metabisulphite solution

25 ml of iodine solution (vii) will be taken in two 250 ml conical flasks (A & B). In A (blank) and B, 25 ml D.D.W. and 25 ml metabisulphite solusion (x) will be added. After keeping flasks in dark for 5 min for reaction, titration will be done with sodium thiosulphate till colour becomes faint yellow. 2 ml starch solution

will be added and titration will be continued until blue colour disappears which was produced due to addition of starch. Titration will be repeated to get constant readings and normality of metabisulphite will be calculated according to the following formula:

Normality of metabisulphite solution

$$= \frac{(A - B) \times N}{Vm}$$

A - Volume of sodium thiosulphate required for blank (flask A)

B - Volume of sodium thiosulphate required for metabisulphite (flask B)

N - Normality of thiosulphate calculated earlier

Vm- Volume of metabisulphite solution taken

Working standard metabisulphite solution in absorbing media

Now solution of metabisulphite will be prepared of such a strength so that 1 ml solution contains 10 µg of SO_2. Total µg of SO_2 in metabisulphite solution will be calculated according to following formula.

Y = Normality of metabisulphite x 32000

Y - Total µg of SO_2 in metabisulphite solution

Now working standard will be prepared in 100 ml absorbing media such 1 ml = 10 µg SO_2

$$Z = \frac{10 \times 100}{Y}$$

Z - the volume of metabisulphite required for dilution to 100 ml with absorbing media.

Calibration of standard curve

Working standard metabisulphite will be pipetted in graduated amounts (such as 0.1, 0.2, 0.3, 0.5, 0.7, 0.9, 1.0, 1.1, 1.2, 1.5 ml which will contain 1, 2, 3, 5, 7, 9, 10, 11, 12, 15 µg SO_2 respectively) into a series of impingers or graduated nessler tubes. Absorbing media will be added into tubes to make volume 10 ml. In the blanks 10 ml only absorbing media will be added. Then 1.0 ml sulphamic acid (ii), 2.0 ml formaldehyde solution (iii), 5.0 ml rosaniline hydrochloric solution (iv) will be added one by one, and after adding each, the solution will be shaken gently. After it the volume of each solution will be maintained with D.D.W. upto 25 ml. After 30 min but before 60 min, the transmittance will be determined at 550 nm in spectrophotometer. A standard curve will be drawn between transmittance and concentration of µg SO_2.

Estimation:

10 ml of sampled absorbing media will be taken in impinger. Then 1 ml sulphamic acid, 2 ml formaldehyde

solution and 5 ml rosaniline will be added one by one and after adding each, the solution will be shaken gently. After 30 min but before 60 min transmittance will be determined at 550 nm in spectrophotometer. In the control (blank) in place of sampled absorbing media non-sampled media will be taken. The ug of SO_2 will be determined by placing of transmittance (%) in calibrated standard curve, the corresponding value (μg SO_2) will be found out. SO_2 $\mu g/m^2$ will be calculated according to following formula:

$$SO_2\ \mu g/m^3 = \frac{\mu g\ SO_2}{\text{volume of air sampled (lit)}} \times 10^3$$

Volume of air sampled = Flow rate of air x time of sampling (minutes)

μg of SO_2/m^3 will be converted into ppm according to following formula:

$$ppm = \frac{\mu g/m^3 \times 22400}{M \times 10^6}$$

M is the molecular weight of the pollutent.

Analysis of nitrogen dioxide (NO_2):-

NO_2 will be estimated by the analytical method. The following reagents will be needed during this process-

Absorbing reagent

4.0 g sodium hydroxide will be dissolved in distilled water followed by the additional 1.0 g sodium arsenite and will be diluted to 1,000 ml.

Sulfanilamide solution

20 g sulfanilamide will be dissolved in 700 ml distilled water. After that 50 ml concentrated phosphoric acid will be added with mixing and will be diluted to 1000 ml.

NEDA solution

0.5 g of NEDA will be dissolved in 500 ml of distilled water.

Hydrogenperoxide solution

0.2 ml of 30% H_2O_2 will be diluted to 250 ml with distilled water.

Standard nitrite solution

Desiccated sodium nitrate will be dissolved in 1000 ml solution with distilled water to make the solution containing 10 µg NO_2/ml.

Standard curve

0.5, 1.0, 1.5, 2.0, 2.5 and 3.0 ml solution will be taken into test tubes followed by the additional 1 ml H_2O_2 solution, 10 ml sulfanilamide solution and 1.4 ml NEDA solution. Side by side the control will also be run.Test tubes will be shaken and allowed to remain at stand for 10 min for about for the colour development.

After that % transmittance (T) will be read at 540 nm at spectrophotometer. A curve will be drawn between concentration and % T.

Estimation :

10 ml of sampled solution will be taken in the test tube. 1 ml H_2O_2 solution, 10 ml sulfanilamide solution and 1.4 ml NEDA solution will be added in the test tube side by side in control absorbing media will be used. Test tubes will be shaken and will be allowed to remain at stand for 10 min for the development of colour. Then transmittance (%) will be taken at 540 nm. And the concentration of NO_2 will be estimated from the standard curve. Now µg NO_2/m^3 will be calculated according to the following formula:

$$\mu g \text{ of } NO_2/m^3 = \frac{\mu g \text{ of } NO_2}{\text{Volume of air sampled (Lits).}} \times 10^3$$

Analysis of ammonia (NH_3):-

Reagents

i) **Absorbing Media**

2.3 ml of concentration H_2SO_4 (18 M) will be diluted in 1 lit distilled water to obtain 0.1N H_2SO_4.

ii) **Nesseler's Reagent (BDH)**

Standard dilution.

iii) **Stock ammonium chloride solution**

8.814 g enhydrous NH_4Cl will be dissolved in 1 lit ammonia free water. One ml of this solution will contain 1.22 mg NH_3.

iv) **Standard NH_4Cl solution**

10 ml of stock NH_4Cl solution will be diluted with ammonia free water to make volume upto 1 lit. One ml of this solution will contain

1 ml = 0.0122 mg NH_3

1 ml = 12.2 µg of NH_3

Calibration of Standard Curve

0.1, 0.3, 0.5, 0.7, 1.0, 1.2, 1.5, 1.8, 2.0, 2.3 and 2.5 ml of standard NH_4Cl solution which will contain 1.2, 3.6, 6.0, 8.4, 12.0, 14.4, 18, 21.6, 24.0, 27.6 and 30.0 µg of NH_3 respectively will be pipetted into marked impingers separately. Then 20 ml absorbing media will be added in each impinger. After that 2 ml Nesseler's reagent will be added in each impinger and in the level of solutions in the impingers will be brought to 25 ml by adding absorbing media. After adding the Nesselar's reagents, impingers will be left for 30 min for colour development. Appearance of yellow colour will indicate the presence of NH_3. In blank all the solutions will be

added except the standard solution of NH_4Cl (iv). After 30 min transmittance in spectrophotometer will be determined at 440 nm of each impinger. Then a curve will be drawn between transmittance (%) and concentration of NH_3.

Estimation :

20 ml sample will be taken in impinger and 2 ml Nesseler's reagent will be added and final volume will be brought to 25 ml by adding more sample. After 30 min the transmittance (%) will be determined in spectrophotometer at 440 nm and by placing the transmittance in standard calibrated curve, the corresponding value (µg of NH_3) will be found out. ug of NH_3/m^3 will be calculated according to following formula:

$$NH_3 \mu g/m^3 = \frac{\mu g\ NH_3}{\text{Volume of air sampled (lits.)}} \times 10^3$$

Analysis of ozone (O_3)

O_3 will be estimated by Alkaline KI method

Absorbing medium

40.0 g of NaOH will be dissolved in distilled water and 10.0 g KI will be mixed to it and the volume will be made upto 1 lit.

Acidifying reagent

5.0 g of sulphamic acid will be dissolved in 100 ml distilled water and after that 84 ml of 85% phosphoric acid will be added to it. The volume of mixture will be brought upto 200 ml with distilled water.

Stock potassium iodate solution

0.15 g KIO_3 will be dissolved in 500 ml of distilled water in a volumetric flask.

Standard (KIO_3) O_3 solution

12.5 ml of stock KIO_3 solution will be taken in 250 ml volumetric flask and its volumes will be brought to 250 ml with double distilled water.

Standard curve

20 ml absorbing media will be taken in to each of the 7 test tubes. After that 0.2, 0.6, 1.0, 2.0, 3.0, 4.0 and 5.0 ml standard O_3 (potassium iodate) solution in test tubes respectively. Then 4 ml acidifying reagent will added with immediate mixing. After that volume of each tube will be made upto 30 ml with distilled water. After waiting for 10-12 min the % transmittance will be read at 355 nm, and the standard calibration curve between % transmittance and corresponding value of O_3 will be prepared. The blank (control) will also run side by side.

Estimation

20 ml sampled solution will be taken into a tube. The blank (control) will have non sampled 20 ml absorbing media. Then 4 ml acidifying reagent will be added in each tubes with immediate shaking. After 10-12 min gap the % tranmittance will be read at 355 nm. The corresponding value of O_3 will be read from standard curve.

Particulate matter :-

In particulate air pollutants, particulate matter fall and suspended particulate matter (SPM) will be determined.

Particulate matter fall :

The amount of particulate matter fall during a fixed period of time will be determined as given below.

Weight of an over dried glass jar (W_1) will be determined. The jar will be placed at the sampling site on a stand 1 m above the ground and will be guarded by grill frame. The upper portion of the jar as well as of grill frame will be kept open to allow dust fall. In jar 1 lit distilled water will be added. This volume will be maintained in jar for one month. After one month jar will be brought to the laboratory and its water will be evaporated by keeping in an oven. Then the weight of the jar

having particulate matter (W_2) will be taken. The total dust fall will be calculated by the following formula.

(A) Total dust fall in one month = $W_2 - W_1$

Generally dust fall rates are expressed in terms of metric tons per square kilometer per month. This will be calculated according to following formula.

Dust fall rate = $4 \times 10^4 \times A / d^2$

A is weight of dust fall in gram in one month

d = diameter of jar

Suspended particulate matter (SPM):

Amount of SPM (g) per meter cube of air will be determined either by High Volume Air Sampler or Handy Air Sampler. The weight of Whatman microfilter paper of grade GF/A of specific size will be taken after putting it into desicicator for 12 h (W_1). Then the filter paper will be fitted in air sampler at the sampling site. Air sampler will be run for 4-8 h depending upon the apparant concentration of SPM. After sampling filter papers will be carefully folded and placed into envelopes and brought to the laboratory. Again weight will be taken after keeping in desiccator for same time (W_2). Suspended particulate matter per cubic meter of air will be calculated according to following formula

Weight of SPM in g (W) = $W_2 - W_1$

$$\text{SPM g/m}^3 = \frac{W}{\text{Volume of air sampled(lit)}} \times 10^3$$

Impact of Ambient Air Pollution on Vegetable Crops

For assessing the effect of air pollutants on vegetable crops, 10 plant samples from each field will be collected from the sites at varying distances from the pollution source. Samples of similar crops will also be collected from the zone demarcated as unpolluted. The samples will be kept in polythene bags and properly labelled. The general crop condition particularly of collection site will be noted (Proforma I). In laboratory the root systems of each plant will be thoroughly washed to remove the soil particles. For plant growth and biomass, length of shoot and roots, weight of fresh and dry plant material will taken for each samples.

Air Pollution Symptoms

Plants collected during the survey (for detecting incidence and intensity of root-knot nematodes;for determining the impact of ambient air pollution on vegetable crops) will be invariably examined closely in the laboratory for detecting symptoms known to be caused by various air

pollutants on plants. If present, symptoms will be matched with the symptoms given in "Recognition of Air Pollution Injury to Vegetation - A Pictorial Atlas" (Eds. S.Jacobson and A.Hill) Air Pollution Control Association, Pittsburg,Pennsylvania, 1970. The details will be noted and photographed.

Physical and Chemical Properties of Soil

In order to assess the effect of air pollution on the characteristics of soil, soil samples will be collected from polluted and unpolluted areas. Soil samples will be collected with the soil augar from surface to 1 feet depth, randomly from the different sites, throughout the polluted areas and unpolluted areas. The soil samples collected in polythene bags and properly labelled will be brought to the laboratory for further testing. The samples will be analysed to determine the following characteristics (Jackson, 1958; Chopra and Kanwar, 1976).

Soil texture :-

International pipette method will be employed to determine the soil texture. 10 g of soil from the treated with H_2O_2 and 0.2N HCl will be dispersed in water using 50 ml sodium oxalate (8 g/lit) as dispersent. Then it will be passed through different mesh sieves. The

percentage of coarse, medium and fine sand will be calculated from the weight of the residues left behind on 25, 72 and 200 mesh sieves. The suspension will then be diluted to 500 ml and transfered to the graduated boiling tube which will be immersed in a constant temperature water bath at 25°C (±1). 10 ml of samples pipetted out carefully at specified intervals of time (4 min and 4 sec, 46 min, 6 h and 5 min) from the depth of 10 cm will be dried and weighed. The percentage of medium silt, coarse silt and clay will be calculated from the weight of the residues.

Water holding capacity:-

For determining the water holding a capacity air dried soil will be crushed in a porcelain mortar and passed through a small sieve of 1.5 m holes. After complete crushing, the coarse particles from the sieve will be mixed with finer particles. The weight of a circular brass box with perforated bottom having filter paper on bottom will be taken. After taking the weight of empty box with filter paper (W_1), it will be filled with soil. The box with soil will be placed in hot air oven for complete drying of soil. After drying again weight will be taken (W_2). Now this will be submerged in a petri dish 1/4 containing water and will be left for 12 h. After this period, the box will be

gently taken out from the petridish, excess water will be allowed to evaporate at room temperature and will be weighed (W_3).

$$\text{Water holding capacity} = \frac{W_3 - W_2}{W_2 - W_1}$$

Soil porosity (Pore space):-

It will be determined by the following formula:

$$\text{Soil porosity} = \frac{100\ (\text{true specific gravity} - \text{apparent specific gravity})}{\text{True specific gravity}}$$

Apparent specific gravity (Bulk density) is the mass of soil per unit volume and porosity of soil is the fraction of soil volume not occupied by soil particles.

Procedure

The weighing bottle of about 50 ml capacity will be weighed first prior to filling with soil, then it will flushed upto the brim tapping the bottle about 20 times and the bottle will be weighed again. After removing the soil the bottle will be filled with water the volume of water needed will be noted. The apparent or bulk density will be obtained by dividing the weight of the soil with volume of the soil.

True specific gravity (Particulate density) of soil

is the arrange density of soil particles. The following method will be followed for its measurement.

Procedure

Already weighed 100 ml specific gravity bottle will be filled with water and weighed again. 10 g. of air dried soil will be taken into a small beaker and few ml of water will be added followed by boiling for short time. Now water will be taken out from the bottle and the soil will be transfered into the bottle. After cooling the bottle at room temperature, it will be filled with water and weighed again.

$$\text{True specific gravity (Particulate density)} = \frac{\text{Weight of soil}}{\text{Weight of water displaced by soil}}$$

After determining the apparent and true specific gravities, soil porosity will be calculated.

Total organic matter or humus :-

The humus content of the soil will be estimated by oxidising or burning the organic matter present in the soil. For this, oven dried soil in crucible, after weighing will be kept in high temperature oven (500°C) for one hour. After that the cooled soil will be again weighed. The difference in the weight of oven dried soil and oxidised or burned soil will be the amount of organic matter present in particular soil sample.

pH :-

Soil pH will be measured by preparing soil suspension in water in the ratio of 1:10. First the sampled soil will be crushed and thoroughly mixed. Then 10 g of soil will be taken with 100 ml double distilled water in 250 ml conical flask and will be agitated for one hour. After it will be filtered with filter paper and filtrate will be used for the measurement of soil pH by glass electrode.

Conductivity :-

For measurement of soil conductivity, 50 g thoroughly crushed and mixed soil sample will be taken, with 100 ml distilled water in 250 ml conical flask. It will be shaken for 20 h or over-night. The suspension will be filtered later on. Conductivity cell will be rinsed with distilled water and then twice with soil water suspension. Cell will be dipped in the solution so that the electrodes will be well immersed. Galvanometer or magic eye of the conductivity meter will be balanced and conductance will be directly read. Temperature will be maintained and will be correlated with the table.

Cation exchange capacity

The capacity of the exchange of equal amount of cation from soil solution is known as cation exchange

capacity (C.E.C.). For knowing the C.E.C. of the soil samples, 10 g of soil will be treated with sufficient amount of 0.1N HCl. After half an hour the soil will be filtered with several washing till the removal of all acidity. Than soil will be kept in saturated solution of KCl for 15 min, then after half an hour, it will again filtered and the filtrate will be titrated with standardized 0.1N NaOH solution.

$$\text{C.E.C.} = \frac{Y \times 100}{10} \text{ meg/100 gm of soil}$$

Y = Volume of 0.1N NaOH used.

Total carbonates and bicarbonates

Total carbonates and bicarbonates will be estimated by the common procedure. 100 gm of soil will be mixed with 50 ml of distilled water and after settling it will be decanted in a conical flask. Then 5 drops of phenophthèline will be added in the flask; the pink colour developed will indicate the presence of carbonates and bicarbonates. Then 0.1N H_2SO_4 will be added till the colour disappears. Then few drops of methyl red indicator will be added in the flask and will be titrated till the colour will not change from yellow to rose red.

The carbonates and bicarbonates will be calculated from the reading.

V_1 = volume of 0.1N H_2SO_4 used with phenophthlein

V_2 = volume of 0.1N H_2SO_4 used in methyl red.

Carbonates:

$$\text{Normality of soil solution } (N_1) = \frac{2\ V_1 \times 0.1}{50}$$

Total carbonates = eq. wt. of CO_3 x normality (N_1)

Bicarbonates:

$$N_2 = \frac{(V_2 - V_1) \times 0.1}{50}$$

Total bicarbonates = eq. wt. x normality of HCO_3

Sulphate :

For estimation of sulphate in the soil samples, 50 ml clear soil extract will be taken into a clean beaker. Then extract will be boiled after adding 5 ml conc. HCl. 5% $BaCl_2$ will be added drop by drop till whole of the sulphate will be precipitated (20 ml solution will be sufficient). Contents of the beaker will be stirred for 30 min and then will be allowed to stand for some time. Then it will be filtered through whatman No.42 filter paper and will be washed with hot water, free from chloride. Precipitate will be dried, ignied and weighed and will be calculated according to following equation:

Wt. of the crucible = W_1

Wt. of the crucible + $BaSO_4$ = W_2

Wt. of $BaSO_4$ = $W_2 - W_1$

Wt. of SO_4 = $\frac{(W_2 - W_1) \times 96}{233}$

% of sulphate in soil = $\frac{(W_2 - W_1) \times 96 \times 500}{233 \times 50}$

Survey and Collection

The survey will be conducted in the air polluted and unpolluted areas around the Thermal Power Plant Kasimpur (Aligarh) during the vegetable growing seasons to assess the incidence and intensity of root-knot disease on some vegetable crops. The types of absorvations to be made and their recording is given in the proforma I.

Root-knot nematodes :-

During the survey about 10 samples (shoot + root) of different vegetables will be collected randomly from fields in different areas in both polluted and unpolluted areas. The samples collected in polythene baga will be properly labelled and brought to the laboratory for further examinations. The root the samples will be thoroughly washed and examined for the presence of galls. Number of galls and egg masses per root system will be counted.

The galls index (GI) and egg mass index (EMI) will be rated according to the following scale.

> 0 = 0, 1 = 1-2, 2 = 3-10, 3 = 11-30, 4 = 31-100 and 5 = greater than 100 galls or eggmasses per root system (Taylor and Sasser, 1978)

To assess the incidence of disease in different area, frequency of occurrence (percentage) of the disease in both the areas on vegetables will be calculated by the following formula:

$$\text{Frequency of occurrence (\%)} = \frac{\text{Number of fields with infection}}{\text{Number of fields surveyed}} \times 100$$

Similarly frequency of occurrence of disease on a particular crop in all the localities will be calculated as follows:

$$\text{Frequency of occurrence (\%)} = \frac{\text{Number of root samples of a crop with infection}}{\text{Number of root samples of the crop examined}} \times 100$$

Frequency of occurrence of the disease and gall and eggmass indices (GI and EMI) will be used as criteria to understand the incidence and intensity of the disease on different crops and in different areas. After indentification of the species persent, occurrence of a species on different crops and in both polluted and unpolluted areas will be also computed. The incidence and intensity

Proforma I:

Studies on root-knot nematodes in relation to environmental pollution.

Department of Botany
A.M.U.,Aligarh (U.P.)

Date _____

Survey No. _____

Collection No. _____

Locality :

Place _____ Town/Vill._____ Distt._____

Crop _____ Age of the crop _____

Abiotic pathogens (Pollutants) _____

Source of pollution _____

Symptoms due to pollutants _____

_____

Biotic pathogens :

Meloidogyne _____

Race _____

GI _____

EMI _____

Symptoms : _____

_____

General crop condition:

No.of samples collected Infected Uninfected

_____ _____ _____

REMARKS:

(MUJEEBUR RAHMAN KHAN)
Research Scholar

of the disease and number of species present in polluted areas will be compared with those of unpolluted areas to assess the impact of ambient air pollution on root-knot disease. For identification of species of root-knot nematodes, pure culturing of the species from collected samples will be done as given below.

Pure culturing :

Pure culutres of the species of root-knot nematode encountered during the survey will be raised and maintained in greenhouse. In order to make pure culture of root-knot nematode, single eggmass inoculation will be made in pots having sterilized soil around the roots of young tomato or eggplant seedlings for each species in glasshouse. Sub-culturing will be done approximatly every 2 to 3 months by inoculating new tomato or eggplant seedlings with atleast 15 egg masses, each obtained from the pure culture in order to maintain sufficient inoculum for further studies.

Preparation of inoculum :

For conducting experiments with root-knot nematodes inoculations of plants will be done by using second stage juveniles (J_2). Second stage juveniles will be obtained by incubating egg masses, collected from roots of tomato or eggplant maintaining pure population of the root-knot nematode species, in sterilized water at 25°C. After 72 h,

number of the hatched juveniles (J_2) per ml will be standardized by counting the ten 1 ml samples and the average number will be used to represent the number of juveniles per ml.

Identification of species and races :-

Samples collected during the survey will be processed for the identification of species and races of the root-knot nematodes. The characteristics of perineal patterns of females will be employed for preliminary identification of the species present in the field samples. Ten to twenty perineal patterns from each field sample will be prepared to identify the species. The root-knot nematodes present in the field samples will be maintained in greenhouse in separate pots as mentioned earlier. The differential host test will then be conducted, which will differentiate the known races of M.incognita, M.arenaria and will also confirm the identity of four most common species (Table 10).

Perrineal pattern method :

To identify the species of Meloidogyne, mature females will be dissected out from large galls on the roots of tomato or eggplants. Ten to twenty perineal patterns will be prepared from each inoculum or sample. Perineal patterns will be viewed under the microscope to

study their characteristics. The species will be identified on the basis of characteristics of perineal patterns (Eisenback et al., 1981).

Differential host test :

North Carolina differential host test (Taylor and Sasser, 1978) will be carried out to determine the species and races of Meloidogyne collected during the survey and maintained in glasshouse. Seedlings of tomato cv. Rutgers, tobacco cv. NC95, pepper cv. California Wonder, peanut cv. Florrunner, watermelon cv. Charleston Grey and cotton cv. Deltapine 16 will be grown in clay pots having sterilized soil in triplicate. Two additional replicates of tomato would be included to determine the time of termination of the test.

After determining the number of juveniles (J_2) per ml, plants will be inoculated with 5,000 J_2/plant per pot. Juveniles will be added to a depression made in the soil at the time of transplanting. Inoculated plants will be kept at glasshouse benches (27-30°C). Fifty to sixty days after inoculations root will be harvested and thoroughly washed with tap water and examined for the presence of galls. Roots with very light infection will be stained with Phloxine B to determine the number of eggmasses. Galls and eggmasses will be counted and GI and EMI will rated on 0-5 scale (Taylor and Sasser, 1978).

Table 10: North Carolina differential host test reaction chart

Meloidogyne species and race	Cotton cv. Deltapine 16	Tobacco cv. NC95	Pepper cv. Californid Wonder	Watermelon cv. Charleston Grey	Peanut cv. Florrunner	Tomato cv. Rutgers
M. incognita						
Race 1	-	-	+	+	-	+
Race 2	-	+	+	+	-	+
Race 3	+	-	+	+	-	+
Race 4	+	+	+	+	-	+
M. javanica	-	+	-(+)	+	-(+)	+
M. arenaria						
Race 1	-	+	+	+	+	+
Race 2	-	+	+	+	-	+
M. hapla	-	+	+	-	+	+

Box indicate key differential host plants

Parentheses indicate that a small proportion of the population attack the host.

After the rating of root system, results will be compared with the differential host test reactions chart (Table 10). This will confirm the identity of the species and will distinguish the races of M.incognita and M. arenaria.

Fecundity :

The number of eggs per eggmass is known as fecundity. It will be measured by shaking vigorously 10 egg masses with 5.25% NaOCl solution. The eggs will be separated from eggmass and collected over 500 mesh sieve. From the sieve the eggs will be transferred into a beaker. 0.33% acid fuchsin (in 25% lactic acid) will be added into 20 to 25 ml of suspension with boiling for 1 minute, for staining the eggs. After cooling, the eggs will be counted and the eggs per eggmass will be calculated to find out the fecundity.

Morphometrical studies :

For the morphometrical studies, the mature females of root-knot nematodes excised from the infected roots will be examined under the microscope and following parameters will be taken into consideration:

Length of the body

Width of the body

Length of the neck

Width of the neck

Length of the stylet

Hight of median bulb

Width of median bulb

Plant Analysis

Analysis of plant samples collected during survey and plants from site and glasshouse experiments will be done for estimating chlorophyll,nitrogen (N), phsophorus (P) and potassium (K).

Estimation of chlorophyll content :-

Chlorophyll contents of the plants samples collected during survey and of the site and glasshouse experiments will be estimated. For chlorophyll estimation 1 g of the interveinal region of the leaves will be ground in 40 ml 80% acetone with the help of mortar and pestle. The suspension will be decanted in Buchner funnel having two whatman paper No.1. Then filtration will be done with the help of suction pump. The residue will be ground thrice adding acetone with 30, 20 and 10 ml of acetone respectively. The suspension will be decanted in buchner funnel and filtered in vaccum. At last mortar and pestle will be rinsed with 80% acetone, transferred in buchner funnel and filtered in vaccum. The filtrate will be transferred in 100 ml volumetric flask and the volume will be made upto

capacity. The transmittance will be read at 645, 663 and 652 nm, at spectrophotometer. The chlorophyll a,b and total chlorophyll will be calculated accordingly by using optical density (O.D.) (by using % transmittance) (Mackinney, 1941).

$$\text{Chl. a in fresh tissue} = 12.7(\text{O.D.}663)-2.69(\text{O.D.}645)\times\frac{V}{100\times W}$$

$$\text{Chl. b in fresh tissue} = 22.9(\text{O.D.}645)-4.68(\text{O.D.}663)\times\frac{V}{1000\times W}$$

$$\text{Total chl. in fresh tissue} = 20.2\ (\text{O.D.}645) + 8.02\ (\text{O.D.}663) \times \frac{V}{1000 \times W}$$

Estimation of NPK

For estimation of NPK leaf samples will be digested as given below.

Digestion of leaf samples

Leaf samples from the survey, site and glasshouse experiments will be digested first according to the following method:

100 mg of oven dried leaf powder will be transferred in 50 ml Kjeldhal flask, than 2 ml of chemically pure H_2SO_4 will be added and flasks will be heated on Kjeldhal assembly for about 2 hours, till the dense fumes has given off and the contents has turned black. Then 0.5 ml of pure 30% H_2O_2 will be added after 15 min of cooling. Now

heating will be done again till the colour is changed into light yellow. It will be heated again for half an hour and after which flask will be cooled for 10 min for getting extract clear. Then 3-4 drops of 30% H_2O_2 will be added dropwise followed by heating for 15 min. After that digested material will be transferred in 100 ml volumetric flask with 3-4 washing and the volume will be made upto capacity. This digested material will be used for estimating N,P,K etc. present in the leaf (Linder 1944; Lundegardh, 1951).

Nitrogen :-

Prior to estimating N content present in the digested material of leaf, standard curve will be drawn by the following procedure.

. 0.236 g of ammonium sulphate will be dissolved in 100 ml of solution, then 0.1, 0.2, 0.3, 0.4, 0.5, 0.6, 0.7, 0.8, 0.9, 1.0 ml solution will be poured in 10 separately test tubes respectively. The volume will then be made upto 5 ml in each test tube by adding distilled water. A control will also be run side by side. After that 0.5 ml Nessler's reagent will be added followed by 5 ml of distilled water. The % transmittance will be read at 525 nm from spectrophotometer on developing yellow arange colour after half an hour. Then a curve will be drawn on graph between concentration and O.D.

Estimation:

10 ml of aliquot (digested leaf material) will be taken in 100 ml volumetric flask and 2 ml, 2.5N NaOH will be added to neutralize the excess amount of acid present. 1 ml of 10% sodium silicate will also be added to prevent turbidity. Then volume will be made upto capacity. 5 ml of aliquot will be taken in three test tubes followed by addition of 0.5 ml of Nessler's reagent with shaking. Then 10 ml volume will be made by distilled water. After waiting for 5 min the % transmittance will be read at 525 nm. Then the O.D. will help in reading the concentration from the standard curve (Linder, 1944).

Phosphorus:-

At first a standard curve will be prepared. Different concentrations of KH_2PO_4 solution ranging from 0.1 to 1 ml will be taken in 10 separate test tubes and the volume of each test tube will be maintained upto 5 ml. Then 1 ml ammonium molybdic acid and 0.4 ml of 1 amino-2-nepthol-4 sulphonic acid in each test tube will be added followed by making the volume upto 10 ml with distilled water. After half an hour % transmittance will be read at 625 nm. Then standard curve will be drawn between concentration and O.D. (Fiske and Row, 1925).

Estimation:

5 ml of aliquot (digested leaf) will be taken in three test tubes, to which 5 ml of distilled water will be added. After that 1 ml of ammonium molybdic acid will be added, with shaking, followed by addition of 0.2 ml 1-amino-2-nephthol 4-sulphonic acid. The controll will also run side by side. Percentage transmittance will be read at 625 nm after half an hour. Concentration will be read from standard graph by using O.D.(Fiske and Rao,1925).

Potassium :-

Standard curve will be made by using the 10 ppm solution of KH_2PO_4. 1 ml, 2 ml ..... 10 ml, 10 ppm KH_2PO_4 solution will be pippetted in 10 ml graduated test tube and the volume will be made upto 10 ml with distilled water. The percentage emission will be read at 740 nm by using flame photometer. The graph will be made between % emission and concentration of KH_2PO_4 in the solution.

Estimation:

1 ml of aliquot (digested leaf material) will be suitably diluted with water. A blank will be run side by side which will have only distilled water. The reading will be taken at 740 nm by flame photometer. Concentration of potassium present will be read by using standard curve.

Physiological Studies

Physiological parameters i.e. rate of water absorption and transpiration will be studied in the plant samples collected from site and glasshouse experiments.

Rate of water absorption :-

For measuring the rate of water absorption by the roots of plant, a wide-mouth bottle with a graduated side tube will be used. The evaporation from side tube will be checked by adding few drops of oil. The rooted plant will be fixed in the mouth of the bottle through a hole in cork. The initial water level in tube will be noted. After a fixed time water level reading will be again recorded. The difference in the water levels will measure the amount of water absorbed by roots.

Rate of transpiration :-

The rate of transpiration will be measured by using Ganong's potometer. In case of small plants, whole of the stem and in case of bigger plants the twigs of equal size will be used, for measuring the rate of transpiration and for comparing with other plants.

Histopathological Studies

Histopathological studies of leaf and root of plant samples collected during the survey and plants from site and glasshouse experiments will be done.

Preparation of leaf peeling :-

Leaf peeling will be prepared by the method given by Ghouse and Yunus (1972). The leaf portion in this method will be boiled in 40 or 60% HNO_3 for 2-3 min. When both the surface will be separated then the leaf peeling will be washed three times in water to remove the acid. After that the peeling will be transferred in 20% KOH for 15 min, which will neutralize the acid. After washing the leaf peeling will be ready for the staining process.

Single staining process:

The washed leaf peels will be kept in 30% alcohol for 10 min, after which it will be transferred in 50%, alcohol for 5 min. Thenafter it will be stained with bismark brown (in 50% alcohol) for 12 h. At the end of this period, the peels will be washed with 50% alcohol, 3 times, with 5 min intervals and will be passed through a series of 70%, 90% and absolute alcohol + xylene and xylene. The peeling will be mounted in Canada balsam. The slides will be ready for examination.

Double staining process:

Leaf peels washed in water will be treated with iron alum (2-3% in water) for 15 min. Thenafter, it will be washed 3 times in water. Dropwise haematoxylene will be

added to water containing the peels. The peels will then be transferred to 30% and 50% alcohol for 10 and 5 min respectively. The peels will then be kept in bismark brown (50% in alcohol for 12 h. At the termination of this period, the same procedure of dehydration, clearing and mounting will be followed as described for single stain.

Histopathology of roots :

For histopathological studies the infected roots will be processed as follows:

The infected roots after being thoroughly washed, they will be fixed in formol-acetic-alcohol (F.A.A.) for 48 h. After fixation the material will be dehydrated by washing the tissue stepwise through increasingly higher concentrations of tertiary butyl alcohol series (Johansen, 1940). After dehydration the material will be embedded in paraffin wax. To ensure complete penetration of wax, the material will be changed twice through warmed paraffin. After cooling the wax, blocks will be made and these blocks mounted on block holder will be trimmed. The sections will be cut about 12-20 u thickness with the help of rotary microtome. The paraffin ribbons containing sections will be mounted with an amount of albumin and glycerine dissolved in water on a clean glass slides. These slides will be put in incubator at 65°C for 1 h to melt the paraffin.

It will be then passed through xylene in order to ensure complete removal of paraffin wax. After this the slides will be passed through descending ethanol series. Staining of sections will be done in safranne and fast green combination (Sass, 1951) and finally mounted in Canada balsam.

Site experiments:

Effect of ambient air pollution on the crop performance and development of root-knot disease on some vegetable plants like tomato, eggplant, okra etc. will be studied in clay pots placed in two net-houses specially fabricated in polluted areas at distances of 1 km and 3 km away from the chimneys of the Thermal Power Plant, Kasimpur. The following treatments will be included for this study.

T_1 = Control (Vegetable crop)

T_2 = Vegetable crop + Meloidogyne incognita

A similar experiment with same treatments will be conducted in unpolluted area (University Farm, 1.5 km away from the Aligarh Muslim University campus)

At the termination of the experiments, the effect of the ambient air pollution will be determined by using following parameter as described earlier.

Shoot length

Root length

Fresh and dry weights of shoot

Fresh and dry weights of root

Number of flowers per plant

Number of fruits per plant

Gall index (GI)

Eggmass index (EMI)

Morphometric studies of mature female of root-knot nematode as described earlier.

Fecundity (number of eggs per eggmass)

Rate of transpiration

Rate of water absorption

Leaf epidermal studies

Chlorophyll and NPK contents of leaf

Glasshouse experiments

Gaseous air pollutants:

Effect of gaseous air pollutants (SO_2, NH_3 and O_3) on the crop performance and development of root-knot disease on tomato, eggplant and okra will be studied in 15 cm clay pots.

Seedlings (3-4-week-old) will be exposed to different doses of the air pollutants in exposure chambers (Fig.A3). Following will be the pattern of treatments for the experiment:

T_1 = Control (without any treatment)

T_2 = Air pollutant

T_3 = M.incognita

T_4 = Air pollutant + M.incognita

T_1 and T_3 pots will be kept in unpolluted air (charcoal filtered air).

At the termination of the experiments, the effect of SO_2, NH_3 and O_3 will be determined by using earlier mentioned parameters.

Particulate air pollutants:

Fly ash originating due to coal burning will be collected and brought to laboratory.

Experiment I : Fly ash will be mixed in soil in different proportion and tomato, eggplant and okra plants will be grown to determine the effect of fly ash on crop performance and root-knot disease development. For this study following treatments will be conducted.

T_1 = Control (soil without fly ash)

T_2 = Fly ash + soil

T_3 = Soil + M.incognita

T_4 = Soil + fly ash + M.incognita

At the termination of experiment to determine the effect of fly ash on plant growth and root-knot development on vegetable crops, previously mentioned parameters will be used.

Experiment II : The collected fly ash will be filled in dust blower upto the mark (2/3 of the blower). Thenafter fly ash will be sprayed on the plants from the seedling stage. Average dust fall per leaf area will be determined by weighing the unit areas of leaf and will be compared with the control at the termination of experiment. For this study following treatments will be made.

T_1 = Control (without fly ash spray)

T_2 = Fly ash spray

T_3 = *M.incognita*

T_4 = Fly ash spray + *M.incognita*

At the termination of the experiment previously mentioned parameters will be used to study the effect of flyash fall on the plant growth and root-knot disease development.

Soil of polluted area :

To assess the effect of soil of air polluted area (Thermal Power Plant, Kasimpur) on plant growth and root-knot disease development, vegetable plants will be grown in clay pots containing autoclaved soil collected from the

polluted areas. For this study following treatments will be made:

T_1 = Control (unpolluted soil)

T_2 = Polluted soil

T_3 = Unpolluted soil + M.incognita

T_4 = Polluted soil + M.incognita

At the termination of the experiment, to determine the effect of soil from air polluted area on plant growth and root-knot disease development on vegetable crops, previously mentioned parameters will be used.

Simulated acid rain :

Vegetable plants grown in clay pots,inoculated and uninoculated with M.incognita, will be sprayed with sulphuric acid of different pH and at different dosages to assess the effect of simulated acid rain on plant growth and root-knot disease development. The following treatments will be included for this study.

T_1 = Control (without treatment)

T_2 = Acid rain

T_3 = M.incognita (without treatment)

T_4 = Acid rain + M.incognita

At the termination of the experiment, the effect of simulated acid rain will be determined by using the previously mentioned parameters.

Heavy metal pollutants:

The effect of different concentrations of heavy metals like mercury, lead, nickel, cobalt etc. will be studied on plant growth and root-knot disease development in artificial treatment conditions. Different concentrations of heavy metals will be added separately in 15 cm clay pots containing autoclaved soil in an amount sufficient enough for homogenous distribution of the metal. Thenafter transplanting or sowing of eggplant and tomato and okra as the case may be will be done. In one set, 2000 freshly hatched juveniles (J_2) of Meloidogyne incognita will also be added to determine the effect on both crop performance and root-knot disease development. The pots will be placed at glasshouse benches and will be terminated after 3-4 months of transplanting or sowing. The treatments will be as follows.

T_1 = Control (without heavy metal)

T_2 = Heavy metal

T_3 = M.incognita

T_4 = Heavy metal + M.incognita

The previously mentioned parameters will be used at the termination of experiments to assess the effect of heavy metal pollution on plants and root-knot nematode disease development.

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